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THE ARNOLD-CHIARI MALFORMATION*

Diagnosis, Demonstration by Intraspinal Lipiodol and Successful Surgical Treatment

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TWO patients who at operation were found to have a malformation of the cerebellum were recently admitted to the Neurological Service at the Massachusetts General Hospital. The rarity of the syndrome, the discovery of the importance of intraspinal lipiodol as a diagnostic method,

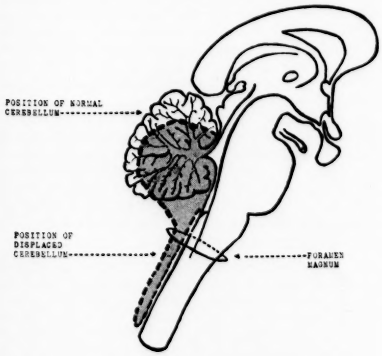

FIGURE 1. *Diagram of Brain Stem and Cerebellum in Sagittal Section.*

and the need of emergency surgery in the treatment of these cases have prompted us to present them in detail.

Arnold,[1] in 1894, and Chiari,[2] in 1895, published original descriptions of this malformation, and since they worked separately, both have been credited with making the first observation. Later, Gredig and Schwalbe[3] published a scholarly account of the embryology and anatomy of this anomaly, and Russell and Donald[4] and Penfield and Coburn[5] have recently discussed the pathologic physiology.

The chief characteristics of the anomaly as given by these different writers are:

It is primarily a congenital malformation of the medulla oblongata and the cerebellum, both of which are herniated through the foramen magnum into the cervical spinal canal (Fig. 1). The part of the cerebellum that is herniated is the inferior portion of both hemispheres, improperly called the "tonsils."

This herniation secondarily leads to the following disturbances: obstruction to cerebrospinal-fluid circulation resulting in hydrocephalus — the cerebrospinal fluid passes from the ventricles into the spinal canal and cannot re-enter the cranial vault, where it is normally absorbed, because of the mass of tissue obstructing the foramen magnum; stretching and injury of the lower cranial nerves; pressure on the spinal cord and the medulla oblongata; and deformity of the cerebellum.

An associated meningomyelocele, together with the Arnold-Chiari malformation, causes most of the patients to die in infancy.

The basal meninges, particularly the pia and arachnoid, are greatly thickened.

Even though this is a rather clear-cut pathologic entity, the Arnold-Chiari malformation is usually discovered at necropsy, not having been suspected during life.

In the last few years, Parker and McConnell[6, 7] and Aring[8] have reported a group of patients

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who during adolescence and early adulthood presented a symptom complex suggestive of a posterior-fossa or high-cervical tumor, but who at operation were found to have the same deformity of the medulla oblongata and cerebellum that Arnold and Chiari had described. These patients did not have a meningomyelocele or other congenital anomalies, and several survived after suboccipital and high-cervical decompression. There was marked thickening of the basal leptomeninges. Whether the deformity of the cerebellum in these cases is also a congenital anomaly is less certain.

The first of our patients is representative of the type of case described by Parker and McConnell; the second illustrates the type described by Arnold and Chiari. We are using the term "Arnold–Chiari syndrome" to designate the aggregate of symptoms and signs by which this malformation manifests itself.

Case Reports

Case 1. J. P. (No. 89142), a 17-year-old schoolboy, was admitted to the Neurological Service of the Massachusetts General Hospital with the following symptoms: suboccipital discomfort, periodic nausea and vomiting, and unsteadiness of gait. Ten months before entry, he sustained a slight head injury when he dived into shallow water. As well as he could remember, his head struck the bottom of the pool, but he was only dazed by the incident and had no difficulty in getting out of the water. The following day, his neck was sore and stiff, and there were pains at the base of his skull. When directly questioned about this symptom, he admitted to having had similar pains for at least 2 years prior to that time. From the date of the injury until admission to the hospital, the headaches became severer and more persistent. Anorexia, nausea and vomiting were frequent symptoms. Three months before admission, the patient fainted and was taken to a local hospital, where a physical examination and x-ray study of the cervical spine disclosed nothing abnormal. He began to have difficulty in walking, owing to stiffness and unsteadiness of his legs. Several times he became choked on food and had trouble drinking water. His speech had always been slurred. Weight loss had amounted to about 25 pounds and was attributed to the nausea and vomiting.

On examination, the blood pressure was 110/94, the temperature 98°F., the pulse 60, and the respirations 16. The patient was a tall, undernourished boy of asthenic habitus. The head and neck were held in a rigid posture but were well centered. He turned his body when looking to either side, and any sudden motion of the head was resisted, because of the pain thus produced. There was very slight but definite blurring of the optic-disk margins, and obliteration of the optic cups. The pupillary reflexes were normal, and ocular movements full. Jaw strength was good, and the sensation over the face was normal. Both sides of the face moved equally in mimicry and laughter. The jaw jerk was hyperactive. No disturbance of either division of the eighth cranial nerve was noted. Two days before operation, the patient was unable to swallow either solid foods or liquids without choking. The sternomastoid and trapezius muscles were of good strength, but the tongue was weak and tremulous, though not atrophied. The speech was somewhat slurred but could have passed for normal. There was an unsustained and at times sustained horizontal nystagmus on both right and left lateral fixation of gaze. There was a slight terminal tremor in doing the finger-nose test, and the muscle tone in the arms seemed diminished. Tests of rapid alternating movements were done well. He tended to walk with a wide base and to deviate to either side. Heel-knee tests were performed with a minimum of unsteadiness. Sensation over the entire body was normal. The tendon reflexes were equal and slightly hyperactive; the abdominal reflexes were absent. There was urinary retention for 24 hours preceding operation.

The blood-cell counts and the urine were normal. Hinton and Wassermann reactions were negative on both serum and spinal fluid. The lumbar puncture showed almost complete dynamic block. Two days later, this was repeated with the same results. The total protein on the cerebrospinal fluid was 78 mg. per 100 cc.; the gold-sol curve was 0012210000, and there were no cells.

X-ray examination of the dural canal after lumbar injection of 5 cc. of lipiodol showed that the lipiodol could be freely moved through the lumbar and dorsal as well as the lower cervical spine. It met with obstruction at the upper edge of the 3rd cervical vertebra, the obstruction being cap shaped. The outline of the cap was hazy and not so distinct as that normally seen in complete obstruction of the spinal canal. No lipiodol was seen to pass beyond the obstruction during fluoroscopic examination of 10 minutes' duration, the patient being examined with his head hanging down at a tilt of 65°. Films taken 10 minutes later showed that some lipiodol had passed by the obstruction and lay in the interpeduncular cistern (Figs. 2, 3 and 4). The amount of lipiodol in the cerebral cavity increased during the next hour, although the patient stayed in horizontal position during this time. A definite upper edge of a tumor mass could not be demonstrated during examinations, which extended over 96 hours, although some lipiodol was constantly visible above the lesion when the patient was in the erect position. The conclusions were as follows:

> The picture is very unusual. The obstruction with cap formation is in favor of a mass in the upper cervical area. There are, however, several features that are unusual for a tumor: the edge is not so distinct as one usually sees in tumor; the lobulation is more pronounced than that seen in tumor; an upper edge of the tumor is not demonstrable, although lipiodol is present above the area of obstruction; and the passage of lipiodol into the cranial vault is comparatively easy for a tumor of this size — there seems to be some kind of valve mechanism allowing the lipiodol to move with comparative ease upward into the skull but not downward from there into the cervical spine.

In spite of these unusual features, the preoperative diagnosis was tumor of the cervical cord, with extension into the posterior fossa.

At operation, performed by Dr. John S. Hodgson and Dr. W. Beecher Scoville, the dura was opened, and what appeared to be normal cerebellar tissue presented itself down to the level of the 3rd cervical vertebra. The arch of the foramen magnum, as well as the bone over the mid-occipital region, was removed, exposing the

cerebellum. The cerebellar hemispheres were small and atrophic and were situated high in the posterior fossa. From the lower portion of the cerebellum, there projected downward two parallel fingerlike processes that overlay the dorsal surface of the medulla and upper cervical cord. These processes were densely adherent and the overlying muscles and skin were closed without drainage.

The patient withstood the operation fairly well, although at the beginning he had a slow pulse and a respiration rate of 10 per minute. For the first hour after the operation, he had generalized muscular tremors. His

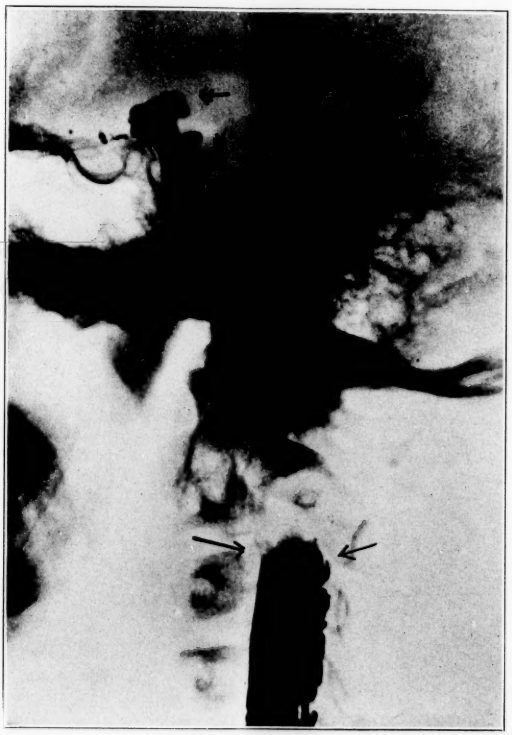

FIGURE 2. *Case 1.*

Lateral view of upper cervical spine and base of skull after the head had been in dependent position for some time. The single arrows show obstruction of lipiodol column at a level that corresponds to lower end of prolapsed cerebellum. The double arrows indicate lipiodol which passed the obstruction and reached the interpeduncular cistern.

both to the spinal cord and to each other. A large vessel with many branches filled the cleft between the two tonsillar projections. By measurement, the fingerlike projections extended a distance of 4 cm. into the cervical canal. An effort was made to pass a catheter between the processes and the cord so as to enter the 4th ventricle and aqueduct, but this proved impossible. Jugular compression was made, and normal spinal fluid oozed slowly from beneath and at the sides of the tonsillar projections of the cerebellum. The dura was left wide open, convalescence proved to be slow and difficult. During the first 2 postoperative days, his respirations were slow (6 to 8 per minute), uneven and stertorous. Repeated inhalations of 5 per cent carbon dioxide in oxygen increased the rate to 14 per minute. Dysphagia was present for several days. Later he developed an acute epididymitis and an acute otitis media. Before discharge, films of the entire spinal column were taken and showed absence of spina bifida. On the 40th postoperative day, the patient was discharged. At that time his headaches

had disappeared, he had no difficulty in swallowing, and his gait was steadier. Otherwise the neurologic findings remained about the same.

During the next 18 months, the patient was followed in the Out Patient Department and continued to improve. He is now working steadily. A lumbar puncture done 3 months after operation showed a partial dynamic block and a total protein of 70 mg. per 100 cc.

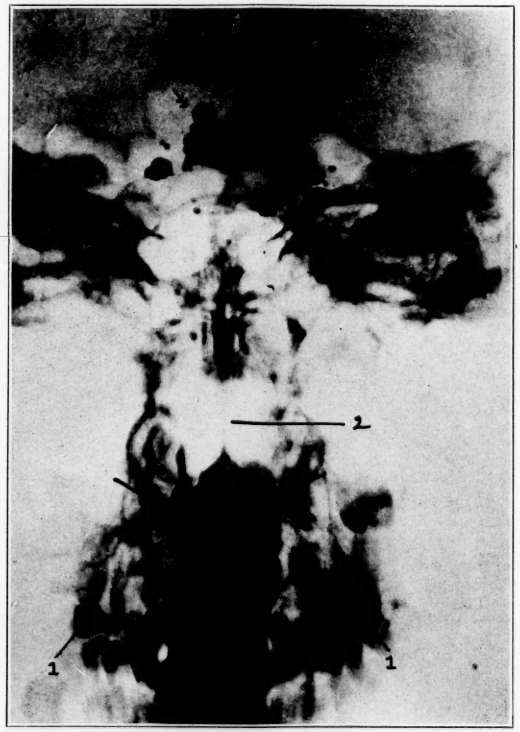

FIGURE 3. *Case 1.*

Posteroanterior view of the skull. The lipiodol column is seen in the upper cervical spine and projected into the area of the teeth. The single arrows indicate the upper edge of the lipiodol column corresponding to the lower edge of the protruding cerebellum. The double arrows indicate lipiodol that passed by the obstruction into the interpeduncular cistern. 1 represents fillings in teeth; 2, the nasal septum; and 3, the frontal sinuses.

CASE 2. A. C. (No. 11486), a 13-year-old girl, had a meningomyelocele at birth; this was operated on during the first day of life, and the sac obliterated. She never developed continent sphincters and did not walk until 5 years of age. Progress in school was excellent, considering her physical handicaps. Several times, she was examined because of urinary incontinence. The bladder was found to be hypertonic, and she once had a transurethral resection of the internal sphincter, with some improvement. She had several operations for equinovarus deformities of the feet. One year prior to entry, she began to have headaches; they were in the occiput and cervical regions, occurring about once a week and lasting several hours at a time, and later became so severe that she was incapacitated. Two months before admission, she vomited for the first time and once fainted for about 10 minutes. For several months, her hands, particularly the left, had become weak. She became so unsteady on her feet that she could walk only with some form of support.

Physical examination showed an intelligent, co-operative girl who seemed rather apathetic. The blood pressure was 116/70, the pulse 80, the temperature 99°F., and the respirations 20. The head was large, measuring 58 cm.

in circumference (occipitofrontal). There was a scar over the spine in the lumbosacral region, and ptosis of both eyelids. Visual acuity, visual fields and optic fundi were normal. There was bilateral weakness of facial muscles, more marked on the right than on the left. Lateral conjugate deviation of eyes to both right and left was very poor. There was coarse, well-sustained synchronous nystagmus with the eyes at rest, which was increased by looking to either side or upward. Upward gaze was normal; convergence was poor. Speech was slurred, and there was slight dysphagia at times. There was weakness of hand grips, all movements of fingers and the flexors and extensors of the wrists. The interossei were atrophied, especially on the left. There was slight scoliosis to the left. Varus deformities of the feet, with weakness in dorsal and plantar flexion, were present. The gait was very ataxic. The only sensory loss was over the 5th lumbar and 5 sacral dermatomes, where all modalities were affected. Tendon reflexes were slightly hyperactive in the arms, but in the legs, knee jerks were diminished and ankle jerks were absent. Abdominal reflexes and extensor plantar responses were absent.

X-ray examination showed a large head and increased convolutional markings, and spina bifida in the lumbosacral region. Lumbar puncture revealed complete dynamic block on jugular compression, but good rise with abdominal pressure and cough. The spinal fluid, which had an initial pressure of 110 mm., was slightly xanthochromic; the total protein was 103 mg. per 100 cc., and the gold-sol curve 0000111000. A pneumomyelogram was unsuccessful.

While we were waiting to inject lipiodol into the spinal

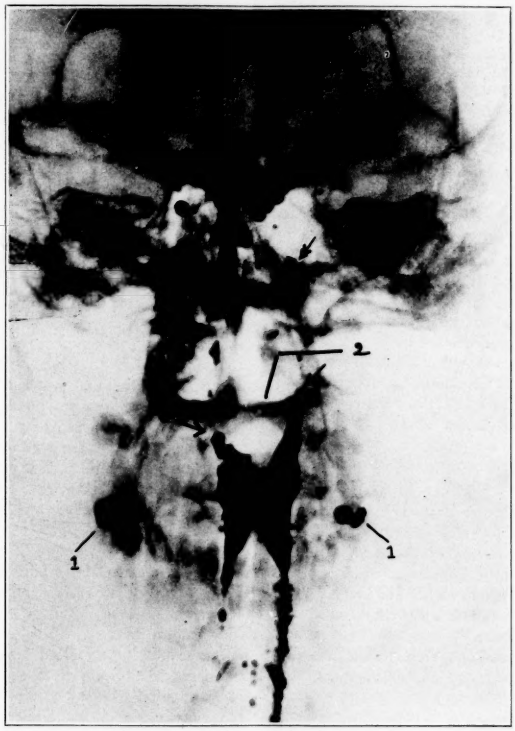

FIGURE 4. *Case 1.*

Anteroposterior view after the patient had been in horizontal position for some time. The single arrows indicate the lower edge of the protruding cerebellum; note its scalloped appearance. The double arrows represent lipiodol in the vault. 1 *represents fillings in teeth;* 2, *the floor of the nasal cavity; and* 3, *the frontal sinuses.*

canal and confirm the diagnosis of meningomyelocele with Arnold–Chiari syndrome, the patient very rapidly became stuporous and soon was deeply comatose. An emergency operation, performed that night, consisted in suboccipital craniotomy and high-cervical laminectomy. A malformation of the hindbrain was found. The cerebellum projected into the cervical canal to the level of the second vertebra, and the medulla was displaced downward so that the cervical roots had to pass upward to where they made their exit through the intervertebral canals. Adhesions of the arachnoid and pia were very dense and were broken whenever possible. Convalescence was uneventful; the patient had no more headaches, the gait was improved, and when the patient was last seen in the Out Patient Department, 6 months after operation, the rest of the neurologic signs were stationary.

Discussion

These cases were clinically similar. In general, the symptoms can be classed under the following headings.

Increased intracranial pressure. Both patients, like many of those reported by Parker and McConnell,[6, 7] had occipital headaches. Usually, the headache is occipital, suboccipital and cervical in location, comes on suddenly and is of several hours' duration, is aptest to occur during the night or in early morning, and sometimes causes a change in state of consciousness, such as fainting. The headache resembles in most respects the cerebellar-tumor headache. Both our patients had vomited with the headaches, and one was nauseated for several months. One patient had slight papilledema. Usually, the papilledema in this syndrome is not so marked as that in cases of cerebellar tumor.

Involvement of several of the cranial nerves. Sometimes the medulla is displaced so far into the spinal canal that the lower cranial nerves must pass through the foramen magnum to enter the cranial vault. Thus they are greatly stretched and often pinched as they go through the foramen magnum. Practically any one or several of the cranial nerves may show signs of injury, especially the lower ones. Our first patient had signs pointing to lesions of the seventh, ninth, tenth and twelfth cranial nerves bilaterally; the other had lesions of the third, seventh, ninth and, questionably, the twelfth.

Compression of the brain stem. The nausea and vomiting are usually attributed to medullary compression, as with cerebellar tumors. Other signs, such as disturbances of conjugate movements of the eyes and vertical nystagmus, are thought to be caused by intramedullary or intrapontine lesions.

Compression of the spinal cord. Both patients had bilateral pyramidal-tract signs; others have had sensory disturbances. Of course, these signs could as well be due to compression of the brain stem. The marked weakness and atrophy of the hands in Case 2 is difficult to explain unless one postulates a small hydromyelia in the cervical cord. Other observers have found such lesions at necropsy.

Cerebellar signs. Symptoms suggesting cerebellar involvement may be the most striking part of the neurologic picture, or may consist of only slight gait disturbance, intention tremor or incoordination of movement.

Several neurologic conditions enter into the differential diagnosis of the Arnold–Chiari syndrome. Depending on whether the cerebellar symptoms, the cord symptoms or the intracranial-pressure symptoms are most marked, one considers cerebellar tumor, syringomyelia and syringobulbia, high cervical-cord tumor, — which extends through the foramen magnum into the posterior fossa, — intrapontine or intramedullary tumor, chronic adhesive arachnoiditis, cerebellar degeneration and platybasia. When the patient exhibits signs of a lesion involving several cranial nerves, the cerebellum, and the brain stem and of increased intracranial pressure, together with significant cerebrospinal-fluid findings (see below), most of the diagnostic possibilities are excluded. The neurologic diseases that should offer most difficulty in differential diagnosis are chronic adhesive arachnoiditis and platybasia.* The latter is a deformity of the occipital bone and foramen magnum, and may produce pressure on the medulla oblongata and adjacent parts of the central nervous system, causing a clinical picture almost identical with the Arnold–Chiari syndrome. It is interesting to note that some degree of platybasia is often found in the Arnold–Chiari syndrome; in all probability it is an associated congenital anomaly of the skull. Nearly all the neurologic diseases considered in differential diagnosis lead to surgical exploration of the posterior fossa or cervical spinal canal; therefore, a diagnostic error is not serious.

Two laboratory procedures were found to be of diagnostic aid: lumbar puncture† and myelography. Lumbar puncture showed a complete block in the dynamics of cerebrospinal fluid, that is, jugular compression caused no rise in spinal-fluid pressure, although coughing, straining and

*This congenital anomaly of the skull, also called "basilar impression," consists in an upward protrusion of the occipital bone around the foramen magnum, and is recognizable because major portions of the axis are above a line that connects the posterior edge of the hard palate with that of the foramen magnum.

†Caution should be taken in doing a lumbar puncture, because a very marked pressure change may cause medullary compression, thus endangering the patient's life. The information gained by this procedure probably justifies its careful use as a diagnostic measure but only after all other procedures are completed. Cisternal puncture is contraindicated.

abdominal pressure gave a prompt rise in pressure. The protein of spinal fluid was significantly increased. These findings are of course not specific for the Arnold–Chiari syndrome but only of a block in the cervical spinal canal. The intraspinal injection of lipiodol gives a very interesting and almost diagnostic picture, since it outlines the bifid projection of the two herniated cerebellar hemispheres.

The following description of the myelographic appearance of the protruding cerebellar "tonsils" seems to be characteristic of the lesion.

A mass within the upper cervical area produces a fairly marked block to lipiodol injected below the lesion. The block is less complete than one would expect from a tumor of similar size, the passage into the vault being less impaired than that in the reverse direction.

The outline of the mass is less distinct than that of tumor, owing to the sloping surface of the soft cerebral tonsils.

The edge of the mass shows lobulation produced by the gyri of the prolapsed cerebellum.

There seems to be a small central incisura within the mass corresponding to the incisura between the two cerebral tonsils.

Some degree of platybasia may be present.

A pneumomyelogram was attempted in Case 2, but was unsuccessful. In the future, this may replace the lipiodol method.

Most neurosurgeons have unsuccessfully operated on these patients. The patient often fails to regain consciousness or dies suddenly some time during convalescence, presumably from compression of the medulla. Probably the wise treatment is to decompress the cervical spinal cord and cerebellum but not to attempt to liberate the nerve structures in the posterior fossa from the arachnoidal adhesions. Any manipulation that causes a sudden change in the spatial relations of these structures may lead to death. Both our patients survived the operation, fortunately, even though more than a simple decompression was done. Preoperative ventricular drainage, as suggested by D'Errico[9] in the treatment of hydrocephalus associated with spina bifida, was not attempted.

Summary and Conclusions

The Arnold–Chiari malformation is a congenital anomaly in which the cerebellum and medulla oblongata are partially displaced into the spinal canal. Certain cases have an associated meningomyelocele or platybasia.

Two cases of Arnold–Chiari malformation are reported, one with and one without meningomyelocele.

The symptoms are divided into five groups: those due to increased intracranial pressure; to involvement of several of the lower cranial nerves; to compression of the medulla oblongata and pons; to compression of the spinal cord; to deformity of the cerebellum.

The differential diagnosis should include such possibilities as high cervical-cord tumor, cerebellar tumor, cerebellar degenerative disease, tumor of the medulla or pons, chronic adhesive arachnoiditis and platybasia.

Lumbar puncture gives confirmatory evidence of the protrusion of the inferior portions of the cerebellar hemispheres into the spinal canal, that is, increased protein and signs of dynamic block.

Roentgenographic examination after intraspinal injection of lipiodol demonstrates a highly characteristic picture of the pathologic process.

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CHROME ULCERATIONS OF THE NOSE AND THROAT*

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CHROMIC acid and its compounds are industrial poisons, and for many years have been known to produce ulceration and perforation of the nasal septum. The first report on the subject was presented by Cumin[1] more than a century ago. He wrote about "chrome holes," which developed in bichromate workers and were chiefly situated on the skin of the hands and arms. He also noted that the septum of the nose was a common site for an ulcerative process.

In 1914, Lehmann[2] made a thorough study of the use of chromates in German industry. He found that the poisoning is local, systemic symptoms being extremely rare.

Experimenting with cats, he found that the inhalation of 8 mg. of chromate per 1000 liters of air was sufficient to produce caustic effects on the mucous membrane of the nasal septum. Death from pulmonary lesions occurred when the animals were subjected to that concentration for three hours a day for a period of five days.

In 1919, Rannelletti[3] described cases among workers in an Italian chemical plant where large quantities of potassium bichromate were used. About half the 69 cases he examined had ulceration of the nasal septum, 6 having complete perforation.

In one of the latest studies reported, Bloomfield and Blum,[4] of the United States Public Health Service and Bureau of Standards, conducted a careful investigation of the health hazards in chromium plating in six plants in this country. They failed to find any evidence of systemic disease in the 23 workers examined.

Workers in the chromium-plating industry, particularly where working conditions are unfavorable, are exposed to the inhalation of a fine brownish spray of chromic acid, which is irritating and corrosive to the mucous membrane of the nose and throat. Chromium plating is accomplished by immersing the object to be plated in an electrolytic tank containing, among other elements, chromic acid. Williams[5] states that the gases evolved from this process, when inhaled, deposit particles of chromium salts on the nasal septum just within and above the nostrils. Ciliary activity is paralyzed, the epithelium becomes corroded, and in time, septal perforation may take place.

It has been my privilege to examine 5 workers employed in a chromium-plating plant. There were 4 men and 1 woman in the group. Working conditions in this plant were very poor, and because of inefficient exhaust ventilation, the atmosphere contained a high concentration of fumes of chromic acid.

There was a similarity in the symptoms of the patients when working over the chromate tanks. An immediate attack of sneezing occurred, associated with epiphora and a profuse watery nasal discharge. Continued close contact produced labored breathing and a choking sensation in the throat. Dizziness, weakness and dull headaches were common symptoms.

After a period of between two and three months, all the patients complained of thick nasal and postnasal discharges. Heavy crusts, which at times were blood stained, were frequently expelled when the patients blew their noses.

Two patients complained of a swollen sensation of the tongue and mouth associated with burning, soreness and dryness. The woman in the group noted an impairment in the sense of taste, and stated that all foods tasted like rubber. If food was eaten on the premises, digestive upsets in the form of nausea and vomiting occurred.

These patients had previously worked in another chromium-plating establishment, where the ventilation was adequate, and none of them had encountered any trouble.

In every case, ulcers were present on one or both sides of the nasal septum. The ulcerations were limited to the cartilaginous portion, and were of various sizes and depths. The ulcers in 3 cases were relatively superficial; there was only a loss of epithelium, covered with a moderately thick dirty-gray scab. In 1 case, the perichondrium had been destroyed, and the erosion had extended to expose bare normal cartilage. One patient had a complete perforation measuring 6 by 4 mm. It was smooth and oval in outline and was situated just above Kiesselbach's area. The mucosa of the turbinates presented a congested appearance. Mucous deposits of a dirty-gray tenacious character were noted on various parts of the turbinates. Crusts were numerous in the anterior nares.

Similar tenacious deposits were found adherent in the postpharyngeal areas. The pharyngeal mucosa was dry and red, and had a glazed appearance. In the patient who complained of a

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rubbery taste, the buccal mucous membrane of the cheeks was thickened and had a milky sheen.

In 2 cases, multiple small ulcers, the edges of which were sharply demarcated, were present on the soft palate, the posterior surface of the tongue and the floor of the mouth. The ulcers were covered with a whitish pseudomembrane, which wiped off easily and did not bleed. They resembled Vincent's ulcers, but smears were negative for spirochetes and fusiform bacilli. The dorsum of the tongue in both these cases was coated, dry and cracked, and many of the papillae were enlarged and red.

All the patients at one time or another had suffered with "chrome holes" on the skin of the hands. These skin ulcers were sluggish in healing, complete healing taking between four and eight weeks.

All patients underwent a complete physical examination by an internist. There was no additional evidence of disease. Blood studies and urinalyses were essentially negative.

Local treatment consisted in cleansing the nose and postnasal space with a weak bicarbonate solution. A bland ointment was applied to the affected parts and sniffed into the nose morning and night, to apply a protective film over the injured parts while regeneration took place.

Chrome ulceration presents a serious industrial problem, so that proper precautions should be instituted at the plant. The danger of dust inhalation can be eliminated by adequate provisions for ventilation, and in this way, the fumes can be diluted down to the point of harmlessness. Since the nose can also be affected by wiping it with contaminated hands, facilities for washing, that is, brushes, soap and individual towels, should be furnished. The mouth and teeth should be rinsed with water before eating and on cessation of work; and before and after each shift, a bit of bland ointment should be sniffed up the nose.

Summary and Conclusions

Five cases of chrome ulceration, 1 with complete perforation of the nasal septum, are presented. Associated lesions in the throat and mouth are described.

Provisions should be made in the chromium-plating trade for the prevention of chrome ulceration, both by mechanical devices for the removal of fumes generated, and by the carrying out of industrial hygienic measures among the workers. Periodic medical examination of workmen should be required, and those showing signs of chrome poisoning should be suspended from the work until the lesions are healed.

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MICROCYTIC, HYPOCHROMIC ANEMIA, ASSOCIATED WITH SPLENOMEGALY AND REFRACTORY TO TREATMENT*

Report of a Case

LEONARD P. ELIEL, M.D.,† AND THEODORE B. BAYLES, M.D.‡

BOSTON

IN the course of observations on the hypochromic anemia of active rheumatoid arthritis, a thirty-nine-year-old Italian woman was encountered with a blood disorder characterized by severe hypochromia and microcytosis resistant to iron therapy, a blood smear showing basophilic stippling, polychromatophilia, target cells§ and marked anisocytosis and poikilocytosis, increased resistance of the red blood corpuscles to hemolysis in hypotonic salt solution, splenomegaly and an elevated serum bilirubin.

Wintrobe et al.[1] have recently reported a somewhat similar blood disorder in five persons over thirty-nine years of age, none of whom, however, had anemia of the severe degree shown by our patient.

The findings in our patient and in the members of her family are given below.

The Patient. J. A., a 39-year-old American-born Italian widow, was admitted to the Robert Breck Brigham Hospital in February, 1940, complaining of migratory, intermittent pain and swelling of the joints of 4 years' duration. Her father and mother were born in Italy and died in old age of a "stroke" and "ulcer of the leg" respectively. The past and system histories were not remarkable, except as presented in the present illness.

For 4 years, the patient had complained of painful swelling in the feet, ankles, knees, fingers and wrists. These symptoms seldom lasted more than a day and left no residual stiffness or disability; they were aggravated by wet weather and had recently become severe enough to interfere with her housework.

The patient stated that she had been "anemic" as long as she could remember. As a child, from about the ages of 7 to 10, she had "spleen trouble," at which time her abdomen was said to have been swollen periodically and to have become normal after the application of "plasters." Her older brother stated that she looked pale at that time, and that the family physician had said she had a very large spleen. These episodes, so far as the patient knew, were not attended by jaundice, chills, fever, nausea or vomiting. She had no more abdominal swelling after childhood. At the age of 8, she had a tapeworm of unknown variety, which was expelled after the administration of small pearl-like pills. She had never had any significant epistaxes, nor had she vomited any blood or coffee-grounds material. Bloody or tarry stools, hematuria, menorrhagia and metrorrhagia were absent, as were episodes of abdominal or back pain, vomiting, chills, fever or jaundice. The patient had never had malaria. There was never a sore mouth or tongue, anesthesias or paresthesias; the appetite was good, and the bowel movements were regular, without diarrhea.

The patient had been a city-welfare case; she ate meat once or twice a week, but few colored vegetables or fruits and no whole-grain cereals, milk or eggs. Her alcoholic intake was very moderate. There was no history of exposure to benzol, lead, hair dyes, arsenic or commercial solvents. She never took cinchophen or other drugs for her joint pains, although she used acetylsalicylic acid occasionally for discomfort.

Physical examination showed a short, pale, somewhat stocky woman who appeared well and was in no great discomfort. The tongue was of normal color and not atrophic. The mucous membranes and conjunctivas were pale. The heart and lungs were normal. The abdomen was normal, except that the spleen was felt to descend on inspiration 3 cm. below the costal margin; its edge was very firm, nontender and smooth. The liver edge could not be felt. There was minimal periarticular thickening about the wrists and knees, and limitation of flexion of both little fingers. A neurologic examination showed normal reflexes and no sensory disturbances.

The blood showed a red-cell count of 4,300,000 with a hemoglobin of 7.9 gm. per 100 cc. of blood and a hematocrit reading of 30 per cent, and a white-cell count of 7600. A differential white-cell count was essentially normal. The blood smear showed marked variation in size and shape of the red cells, and there were a large number of microcytes, some well filled with hemoglobin. Many larger cells were markedly hypochromic. There were numerous pear-shaped and band-shaped, tailed, elliptiform and target cells. There were occasional cells showing polychromatophilia, and other cells with basophilic stippling. Smears at different times during hospitalization showed no normoblasts, but they were occasionally present (0 to 4 per 100 leukocytes) after the injection of epinephrine. Reticulocyte counts averaged 1 per cent. The platelets were normal to increased in number. Subsequent examination revealed that the color index was 0.51, the mean corpuscular volume 61.2 cubic microns, the mean corpuscular hemoglobin 15.9 micromicrograms and the mean corpuscular hemoglobin concentration 25.9 per cent. The serum bilirubin was 1.3 mg. per 100 cc., with an indirect van den Bergh reaction. A fragility test showed hemolysis beginning at 0.44 per cent saline solution, becoming definite at 0.28 to 0.32 per cent and complete at 0.12 per cent—a control showed beginning hemolysis at 0.44 per cent, becoming definite at 0.38 to 0.40 per cent and complete at 0.28 per cent. A test for urine urobilinogen with Ehrlich's reagent in serial dilutions of urine showed no urobilinogen beyond a dilution of 1:4 (control 1:8). Examination of wet films again showed the many bizarre forms seen in the smear, with many unusually flat irregular cells; there were no spherocytes. An anaerobic preparation failed to develop sickling of the red cells. No malarial parasites were seen.

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†Formerly, student house officer, Robert Breck Brigham Hospital.

‡Research fellow, Robert Breck Brigham Hospital.

§Within the usually clear central area of the erythrocyte, there is a small island (bull's-eye) of hemoglobin, hence the resemblance to a target.

The serum protein, albumin-globulin ratio, nonprotein nitrogen and fasting cholesterol were all normal. An analysis of fasting gastric contents showed 5 units of free and 6 units of total acid, after histamine, 82 units of free and 85 units of combined acid. The basal metabolic rate was −23 per cent, and following 2 weeks of therapy with 0.1 gm. of thyroid daily was −12 per cent. Seven urine examinations showed nothing abnormal. Six stool examinations were negative for occult blood, and three revealed no parasites or ova.

During the patient's stay a sternal bone-marrow biopsy was performed, and through the courtesy of Dr. J. Beach Hazard, reported as follows:

> The marrow is of markedly increased cellularity, only about 10 per cent of fat cells being evident per field, as compared with the normal. Scattered megakaryocytes are present and appear in about normal number. The increase in cells is due for the most part to an increase in nucleated erythrocytes, either pyknotic or normoblastic. There is no increase in stem cells or megaloblasts. A differential count based on 600 cells is as follows: stem cells, 1 per cent; blasts, 10 per cent; nucleated red cells, 69 per cent (21 per cent normoblasts, 48 per cent pyknotic nucleated red cells); polymorphonuclear neutrophils, 10 per cent; polymorphonuclear eosinophils, 3 per cent; eosinophilic myelocytes, 5 per cent; and neutrophilic myelocytes, 2 per cent. Diagnosis: marked erythrocytic hyperplasia of normoblastic type, with a large increase in nucleated erythrocytes.

X-ray study showed bone changes consistent with mild rheumatoid arthritis. A skull plate was normal. There was no rarefaction or thinning of the cortex of the long bones. Thick and thin barium meals showed no gastric or esophageal varices or gastric or duodenal ulcer. A plain film of the abdomen showed the spleen to be enlarged.

During the patient's hospital stay and until August 13, 1940, she was given in addition to thyroid, as above, ferrous sulfate, brewer's yeast, cevitamic acid and haliver oil. She received the routine orthopedic care, including postural exercises, occupational therapy, wax to her hands, Castex wristlets and beaver board under her mattress. Her course was uneventful; she was free from complaints soon after her arrival and was discharged in March. Since discharge, she has continued the therapy given during her hospital stay, but the blood findings have remained essentially unchanged from those at discharge (Table 1).

TABLE 1. *Summary of Findings in J. A. and her Family.*

FINDINGS	J. A.				M. F. (DAUGHTER)	ILL. A. (DAUGHTER)		F. Y. (BROTHER)	M. Y. (BROTHER)	C. H. (SISTER)
	2/16/40	3/11/40	4/25/40	8/13/40	4/25/40	4/25/40	8/13/40	4/30/40		6/26/40
Physical examination										
Jaundice	0	0	0	0	0	0	0	0	0	0
Splenomegaly	++	++	++	++	0	0	0	0	0	0
Urobilinuria	0		0							
Blood examination										
Erythrocytes (millions cu. mm.)	4.3	4.7	4.9	4.7	5.4	5.3	4.5	5.6	5.0	4.2
Hemoglobin (gm. 100 cc.)	7.9	7.5	7.3	7.3	12.2	12.3	11.7	13.1	95%	12.0
Hematocrit (%)	30	28	29	27	41	40	37	45		40
Color index		0.50	0.51	0.50	0.71	0.75	0.83	0.75		0.90
Mean corpuscular volume (cu. microns)		61	58	56	73	74	83	81		95
Mean corpuscular hemoglobin (micromicrograms)		15	15	15	22	22	26	23		28
Mean corpuscular hemoglobin concentration (%)		25	25	27	30	30	31	28		30
Van den Bergh (mg./100 cc.)		1.3	1.1	1.2	1.1	0	0			0.3
Icteric index of plasma				11						
Reticulocytes (%)		1.2	1.3	1.8	1.0	0.8	1.0			
Abnormal erythrocytes										
Hypochromia	+++	+++	+++	+++	+	+	+	+		0
Anisocytosis	+++	+++	+++	+++	+	+	±	0		0
Poikilocytosis	+++	+++	+++	+++	±	+	±	0		0
Target cells	++	++	++	++	0	±	±	0		0
Polychromatophilia	++	++	++	++	±	0	0	0		0
Stippling	+	+	+	+	0	0		0		
Nucleated red cells	0	0	0	0	0	0	0	0		
Spherocytes	0	0	0	0	0	0	0	0		
Fragility test:										
Hemolysis begins		0.36	0.44	0.42	0.44	0.44	0.38	0.44		0.40
Hemolysis definite		0.32	0.28	0.38	0.42	0.42	0.34	0.42		
Hemolysis complete		0.19	0.12	0.10	0.30	0.30	0.22	0.34		0.30

In the above table, the following abbreviations are used: 0, absent; ±, rare; +, present; ++, moderate; +++, marked.

The Patient's Children. M. F., a 21-year-old married daughter, had always been well. A stained blood smear showed slight variation in size of the red cells, with occasional tailed or band forms. A very rare macrocyte was seen, in addition to slight polychromatophilia. The remainder of the findings are shown in Table 1. This patient did not have her mother's blood disorder.

Jul. A., a 17-year-old daughter, had always been well. Her blood examination revealed a red-cell count of 5,370,000 with a hemoglobin of 12.3 gm. per 100 cc. and a hematocrit reading of 40 per cent. The color index was 0.75, the mean corpuscular volume 74.5 cubic microns, the mean corpuscular hemoglobin 22.8 micromicrograms and the mean corpuscular hemoglobin concentration 30.7 per cent. The smear showed slight but definite variation in size, and rare microcytes and band-formed, elliptiform, tailed and pear-shaped cells consistent with the degree of secondary anemia present. One target cell was seen in counting 200 white cells. The reticulocyte count was 0.8 per cent. There was no polychromatophilia or basophilic stippling. The differential leukocyte count was normal. A serum bilirubin was normal. A fragility test showed hemolysis beginning at 0.44 per cent saline solution, becoming definite at 0.42 per cent and complete at 0.30 per cent; a control blood showed identical hemolysis. Skull roentgenograms were normal.

Because of the blood smear and the degree of anemia, a course of iron therapy was given to discover if the changes in the stained blood smear were those secondary to a simple iron-deficiency anemia or related to the blood disorder of her mother. For 6 weeks, this girl took 0.7 gm. of ferrous sulfate daily, and examination of her blood at the end of that time pointed toward a simple iron-deficiency anemia (Table 1); there were fewer larger cells, better filled with hemoglobin, and the stained blood smear had changed slightly but definitely toward normal.

The Patient's Brothers. F. Y., a 54-year-old tavern keeper, had always been well. A stained blood smear was normal, and other laboratory studies (Table 1) were all within normal limits.

M. Y. had died, at the age of 49, of epidermoid carcinoma of the lung. Except for mild asthma and emphysema preceding his terminal illness, a gonorrheal infection and a head injury, his history was not remarkable. Only an admission blood was recorded during his terminal illness; it showed a red-cell count of 5,050,000 with a hemoglobin of 95 per cent (Tallqvist), as shown in Table 1. An autopsy was limited to the operative wound, and no bone-marrow samples were studied.

The Patient's Sister. Mrs. C. H., aged 52, had always been well; her blood was normal.

Discussion

From examination of the data, it can be seen that the patient suffered from a marked microcytic hypochromic anemia with a majority of microcytic hypochromic cells and, in addition, an impressive minority of macrocytic hypochromic cells with an irregular distribution of hemoglobin. There was evidence of marked bone-marrow activity, as indicated by the presence of polychromatophilia, basophilic stippling, normoblasts and young white cells. The increase of the young red-cell elements after the injection of epinephrine, and the bone-marrow biopsy showing a profusion of normoblasts, confirmed this impression. There was a chronic mild bilirubinemia. The increased resistance of the red cells to hemolysis in hypotonic saline solution was consistent with the degree of microcytosis. In addition, the interesting history of splenomegaly in childhood and the long-standing symptomless anemia should be noted. The anemia was resistant to therapy, including large amounts of iron and extrinsic factors.

The family of the patient could not be shown to have this blood disorder.

The patient's blood disorder differed from Mediterranean anemia (Cooley's erythroblastic anemia[2]) only in lacking erythroblasts in the circulating blood and in roentgenographic changes in the bones. The latter may well be related to bone-marrow hyperplasia in growing bone.

This condition differs from familial microcytic anemia[3] because of absence of the disorder in other members of the family and the absence of skull changes with roentgen-ray study. In addition, our patient had splenomegaly, a permanently elevated serum bilirubin and a more abnormal stained blood smear.

The disorder appears to be similar to some of the cases described by Wintrobe et al.[1] It is also similar to the condition described by Dameshek[4] as "target-cell anemia," except for the absence of bone changes.

Summary

A case of hypochromic microcytic anemia, associated with splenomegaly and subclinical jaundice resistant to therapy, is presented, and its differences from previously described idiopathic anemias summarized. The erythrocyte changes bear marked similarity to those found in Mediterranean anemia in children.

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NEW HAMPSHIRE MEDICAL SOCIETY

PROCEEDINGS OF THE ONE HUNDRED AND FIFTIETH ANNIVERSARY

House of Delegates, May 12, 13 and 14, 1941

THE House of Delegates convened at the Hotel Carpenter, Manchester, on May 12, 1941, at 7:30 p.m., with Speaker Robert O. Blood, of Concord, presiding.

The following members answered the roll call:

The President, *ex-officio*
The Vice-President, *ex-officio*
The Secretary-Treasurer, *ex-officio*
Lyall A. Middleton, Plymouth
Richard W. Robinson, Laconia
Francis J. C. Dube, Center Ossipee
W. J. Paul Dye, Wolfeboro
Norris H. Robertson, Keene
Walter F. Taylor, Keene
Leslie K. Sycamore, Hanover
Everett C. Campbell, Woodsville
Frederic P. Lord, Hanover (alternate for Willard A. Bates, Littleton)
Deering G. Smith, Nashua
Henry O. Smith, Hudson (alternate for Charles H. Cutler, Peterboro)
Clarence E. Dunbar, Manchester
George V. Fiske, Manchester
George C. Wilkins, Manchester (appointed as alternate for Luther A. March, Nashua)
Warren H. Butterfield, Concord
Charles H. Parsons, Concord
Herbert B. Messinger, Franklin (alternate for Robert J. Graves, Concord)
Martha I. Boger-Shattuck, Portsmouth
James Sanders, Rye
Frederick S. Gray, Portsmouth
Edna Walck, Dover
Albert E. Barcomb, Rochester
Henry C. Sanders, Jr., Claremont
William M. Prince, Newport

The Speaker declared a quorum present, and appointed the Credentials Committee as follows: Drs. Dunbar (chairman), Dube and H. C. Sanders, Jr. Dr. Dunbar reported that the credentials were in order.

The Speaker appointed the Committee on Officers' Reports as follows: Drs. D. G. Smith (chairman), Sycamore and Robertson. He appointed the Committee on Memorials and Communications as follows: Drs. Parsons (chairman), Beaudoin and Middleton. To the Committee on Nominations he appointed Drs. Robertson (chairman), Robinson, Dunbar, Gray and Walck.

The secretary-treasurer, Dr. Carleton R. Metcalf, presented his report.

Report of the Secretary-Treasurer

The following report for 1940 is submitted:

MEMBERSHIP, DECEMBER 31, 1940

PAID

Belknap County	32
Carroll County	14
Cheshire County	31
Coos County	40
Grafton County	61
Hillsborough County	127
Merrimack County	71
Rockingham County	55
Strafford County	28
Sullivan County	19
Not in county society	5
	483

UNPAID

Affiliate members	27	
Honorary members	11	
		38
Total		521

The total membership on December 31, 1939, was 526.

FINANCIAL STATEMENT

RECEIPTS

January 1, 1940—balance forward	$1,210.84
Belknap County	192.00
Carroll County	90.00
Cheshire County	192.00
Coos County	276.00
Grafton County	372.00
Hillsborough County	781.00
Merrimack County	414.00
Rockingham County	336.00
Strafford County	180.00
Sullivan County	120.00
Cash received at annual meeting	42.00
Members not in county societies	30.00
Receipts from Bartlett Fund	2,000.00
Benevolence Fund (Women's Auxiliary)	225.00
New England Journal of Medicine subscriptions	6.00
Refund, Cancer Commission	5.68
	$6,472.52
Net receipts from 1940 annual meeting, deposited 3/14/41	248.42
	$6,720.94

EXPENDITURES

New England Journal of Medicine (journals)	$614.73
New England Journal of Medicine (full subscriptions)	6.00
New England Journal of Medicine (transactions)	523.68
New England Journal of Medicine (half-tone cut)	1.28
Carleton R. Metcalf (salary)	400.00
Bridge and Byron (printing)	88.87
Envelopes, stamps and pamphlets	102.31
Eagle and Phoenix Hotel Co. (committee lunches)	16.15
Robert O. Blood (telegrams and telephone calls)	21.40
Robert O. Blood (clerical work, 1938–1940)	150.00
Women's Auxiliary	100.00
Benevolence Fund	477.00
Frank J. Sulloway (retaining fee)	100.00
Florence McCann (Committee on Education and Hospitals)	14.86
The Barwood Press (Committee on Education and Hospitals)	6.75
Dartmouth College (Committee on Education and Hospitals)	4.41
The Robbins Company (medals)	45.42
Concord Photoengraving Co. (half-tone cuts)	36.56
Frederic P. Lord (money allotted to General Fund)	1,000.00
Ernest M. Hopkins (gift, Dartmouth College)	2,000.00
Gray Service, Roosevelt Hospital (Dr. Mills's expenses at annual meeting)	39.00
C. P. Rhoads (expenses, annual meeting)	23.20
George C. Wilkins (Cancer Committee)	50.00
Leander P. Beaudoin (dues collected at annual meeting)	7.00
Deering G. Smith (dues collected at annual meeting)	35.00
Frederick S. Gray (dues collected at annual meeting)	7.00
Roy F. Plummer (primary expenses at Laconia)	100.00
John R. Perley (primary expenses at Laconia)	50.00
Deering G. Smith (expenses A. M. A.)	68.00
Madeline A. May (stenographer at annual meeting)	236.43
James B. Woodman (telephone calls)	12.55
Reliable Wiring Co. (lamp for index file)	14.24
National State Capital Bank (service charge)	1.00
	$6,352.84
Balance in check book, April 5, 1941	119.68
	$6,472.52
Net receipts from annual meeting deposited 3/14/41	248.42
	$6,720.94

The Society is in good financial condition, with a balance of $119.68 in the bank on January 5, 1941. The Benevolence Fund on the same date amounted to $2944.00; of this amount the principal is $2594.19, and the accrued income, $349.81. During the past year we have received $225.00 for this fund from the women's auxiliaries of the following counties:

Strafford	$50.00
Coos	10.00
Merrimack	25.00
Rockingham	40.00
Hillsborough	100.00

We gave $100.00 to Mr. Frank J. Sulloway as a retaining fee, and $100.00 to the Auxiliary to cover, in part, the expenses of its meeting last May.

Two officers of the Society have died during the past year: Osmon H. Hubbard, of Keene, and Fred E. Clow, of Wolfeboro. Both were members of the Advisory Committee on Jurisprudence. Dr. Hubbard was a former president of the Society, and Dr. Clow a former speaker of the House of Delegates.

The President chose David W. Parker, of Manchester, for anniversary chairman and appointed Harry O. Chesley, of Dover, to membership on the New England Medical Council. The value of the New England Medical Council seems to me highly questionable. It has not had a meeting for years, and two or three years ago when we tried to get the different New England states to hold a meeting, Massachusetts was the only state that responded. It seems rather a futile gesture to appoint members to the council annually.

A year ago the House of Delegates made the following recommendations, which have been carried out:

Fifty dollars was given to the Cancer Committee for its work.

The members of the Society were urged to accord moral and financial support to the National Physicians Committee for the Extension of Medical Service.

A Committee was appointed to co-operate with the University of New Hampshire in celebrating its seventy-fifth anniversary.

I requested the chairman of the Committee on Officers' Reports and the chairman of the Committee on Memorials and Communications to send to each delegate an agenda of important matters to be taken up by delegates at this meeting.

Two thousand dollars was sent to Dartmouth College for use of the Medical School.

I conveyed to our United States senators the resolutions of the Society pertaining to the Wagner Act.

I conducted a postal-card ballot concerning the elimination or retention of round-table conferences at our annual meetings. The results were as follows:

Do you prefer to return to the former plan of having members read set papers? Yes, 44. No, 129. No answer, 10.

If we return to the former plan, will you be willing to write and read a paper? Yes, 54. No, 57. No answer, 64.

Prefer present plan but will write a paper if former plan adopted: 26.

Prefer former plan and will write a paper if former plan adopted: 25.

Prefer neither plan but will write paper if former plan adopted: 3.

There has been very little important medical legislation in the present session of the Legislature. I have appeared before a committee only once, to oppose the Federal Narcotic Bill, which contains a great deal of red tape and which would compel the State, at considerable expense, to carry on the work that is now being adequately done by the federal government. The bill was defeated.

The State Board of Health has requested us to appoint a Committee on Public Health to confer with them from time to time on matters in which they wish the opinion of the Society.

I shall be glad to have you instruct me in regard to

the action that I should take concerning doctors who are now or who will be in the United States Army. Should they be continued as members of the Society without payment of dues or should they be dropped from the rolls?

The President and I have had several meetings with General Bowen in regard to Selective Service. General Bowen requested us to choose doctors in various parts of the State to carry on the work of examining the draftees. We selected 176 doctors, and I am happy to say that every one of them agreed to serve without pay.

CARLETON R. METCALF, *Secretary-Treasurer*

Dr. Metcalf added that several other things should be mentioned: first, a communication from the Hartford Accident and Indemnity Company to the effect that, effective May 1, 1941, the annual rate for malpractice liability insurance for members of the Society had been reduced from $40.00 to $37.50 for the standard coverage of $5000 to $15,000; secondly, that $1000 had been transferred from the General Fund to the Benevolence Fund; and, thirdly, that the expenditure of $150 for political work in Belknap County seemed justified to the officers of the Society.

The report was accepted and was reported on by Dr. D. G. Smith, of the Committee on Officers' Reports. Dr. Smith spoke of the fact that several committee chairmen had failed to forward their reports to his committee in time for copies to be sent to the delegates; after some discussion, it was the consensus of the delegates that the chairmen should observe more strictly the regulation requiring the filing of reports by April 15.

Dr. Smith recommended that Article XII of the constitution, relative to the New England Medical Council, be deleted. (Action on this proposed amendment will be taken in 1942.)

Dr. Smith recommended that a standing committee, the Committee on Public Health, be appointed; after considerable discussion it was voted to table the question until the following day.

Dr. Smith recommended that the dues of members on extended active duty with the military and naval services of the United States be remitted for that calendar year in any part of which they are on active duty; it was so voted.

Because of the amount of business to be transacted, it was voted to omit the reading of the councilors' reports.

Reports of Councilors

BELKNAP COUNTY

The Belknap County Medical Society held six meetings from November to April inclusive. The meetings were very well attended, some members driving over fifty miles to attend. As usual a dinner was served at 6:15, followed by the program. The Women's Auxiliary met at the same time and place.

The society has thirty-seven members.

C. S. ABBOTT

CARROLL COUNTY

The Carroll County Medical Society has held its meetings at Center Ossipee as usual. The Auxiliary has held its meetings at the same time and place.

Our numbers are small, since our membership comprises but sixteen names; however our spirit is excellent and our meetings well attended.

C. E. SMITH

CHESHIRE COUNTY

The Cheshire County Medical Society held two well-attended meetings during 1940. At each meeting outside speakers presented papers of interest. These papers were given in such a way that much discussion followed. Discussions always stimulate interest and add to the value of such gatherings.

Three new members have been added to the society during the past year. This addition, however, did not swell our membership, since we lost three members by death: Dr. Hubbard, of Keene; Dr. Goldsmith, of Winchester; and Dr. Sheldon, of Troy. Dr. Goldsmith and Dr. Sheldon were fairly recent members, but by Dr. Hubbard's death the society lost one of its oldest members. Though quiet and unassuming, Dr. Hubbard had the faculty of accomplishing whatever he undertook. His activities in county and state medicine were many — to mention but a few, he had been president of the county society, president of the New Hampshire Medical Society, president of the staff of the Elliot Community Hospital and state pathologist.

JOHN J. BROSNAHAN

COOS COUNTY

We had no fall meeting this year. Our annual meeting was held in Berlin on April 26, 1941. The guest speaker was Dr. Henry Faxon, from the Massachusetts General Hospital, who gave us a very interesting talk on the subject, "Diagnosis and Treatment of Common Peripheral Vascular Diseases."

The society voted to let the Farm Security Administration function in our county.

We have forty-one members now, having lost two by removal and one by death. On the whole, our society is in a healthy condition.

We feel the loss of Dr. Homer Marks, who died on April 11, 1941, following an accident.

RICHARD E. WILDER

GRAFTON COUNTY

The annual business meeting of the society was held at the Hitchcock Hospital, Hanover, on October 30, 1940. President Ezra A. Jones, of the New Hampshire Medical Society, and Deering G. Smith, chairman of the New Hampshire Committee on Medical Preparedness, were present and led in the discussion of problems of medical preparedness. Dr. R. C. Tanzer, of Hanover, presented an interesting paper, "Special Problems in Plastic Surgery."

The spring meeting was held at the Hitchcock Hospital, Hanover, on March 28, 1941. It was voted to ap-

prove the plan of the Metropolitan Casualty Insurance Company for group disability insurance for members of the society. A sound motion picture, "Conception and Contraception," furnished by the Ortho Products Company, was presented.

The society has contributed to the support of the National Physicians Committee and has also contributed an emergency medical kit to the Medical and Surgical Relief Committee for Britain.

ARTHUR W. BURNHAM

HILLSBOROUGH COUNTY

The Hillsborough County Medical Society has had two meetings since the last annual meeting of the New Hampshire Medical Society.

On October 29, 1940, we met at the Manchester Country Club and the meeting was presided over by Dr. J. S. Black, of Nashua. At that time, six new members were admitted to the society; since then, two have been lost by death. At the business meeting a very interesting talk was given by Dr. Deering Smith, the New Hampshire delegate to the American Medical Association, on the subject, "Medical Preparedness." This was followed by much discussion. Remarks were made by Dr. Ezra Jones, president of the New Hampshire Medical Society, calling to our attention the fact that 1941 was the one hundred and fiftieth anniversary of the New Hampshire Medical Society. After luncheon, a paper was presented by Dr. Lyman G. Richards, of Boston. The topic of Dr. Richards's interesting paper was "Practical Bronchoscopic Diagnosis and Therapy," illustrated. Another paper of interest was by Dr. Joe Vincent Meigs, of Boston, and was titled, "Symposium on Vaginal Bleeding: Amenorrhea, dysmenorrhea and abnormal bleeding."

Our spring meeting was held in conjunction with the New Hampshire Surgical Club at the Nashua Country Club on April 29, 1941, with over a hundred physicians attending; Dr. George T. Sheehan, president of the society, presided.

The scientific program of this meeting included surgical clinics at St. Joseph's and Memorial hospitals and papers on the following subjects: "The Treatment of Toxemias of Pregnancy," by Dr. Marion Fairfield; "Peritonitis," by Dr. Loren F. Richards; "Middle-Ear Infection," by Dr. Raymond Marcotte; and "Large Inguinal Rings as a Bar to Employment," by Dr. Richard Mulvanity. During the afternoon Dr. Joseph W. O'Connor, of Worcester, Massachusetts, presented a very interesting paper, "Spontaneous Rupture of the Uterus: Its diagnosis and treatment." He was followed by Dr. John M. Birnie, of Springfield, Massachusetts, who gave an excellent presentation on the subject, "Large Inguinal Rings and Potential Hernias." Both of these were discussed by members of the society and the club, and then followed a motion picture, "Vitamin B Deficiencies and Their Treatment," shown by E. R. Squibb and Company.

Of the 179 physicians in Hillsborough County, 140 are members of the Hillsborough County Medical Society. Several new members were admitted at this meeting. Four of our members, Dr. R. E. Fiske, Dr. G. J. Kapopoulas, Dr. R. M. Bremner and Dr. D. L. Hallisey, are on active duty with the National Guard. There are eight medical reserve officers in the county, of whom Dr. M. O. Goodman, Dr. P. R. Hamel, Dr. R. E. Lapointe and Dr. R. R. Rix are on active duty for a year.

The accident and health insurance plan which was proposed to our members at the last meeting and approved by them has gone into effect, over half the eligible members taking out policies.

CLARENCE O. COBURN

MERRIMACK COUNTY

A summer meeting was held at Franklin at the Country Club on July 10, 1940. About twenty members attended. Dr. Levine, of Bristol and Concord, gave a paper on superstitions relating to medical treatment in the early New England period. A local county defense committee, consisting of Dr. McQuade, of Franklin, Dr. MacLean Gill, of Concord, and Dr. Warren Butterfield, of Concord, was elected.

At the fall meeting, held on October 2, 1940, at the Eagle Hotel, Dr. Deering G. Smith discussed the defense program and the part the medical profession will have in it.

On January 8, 1941, the following officers were elected: president, Dr. MacLean J. Gill; vice-president, Dr. Edward Putnam; secretary-treasurer, Dr. Warren Butterfield. Mr. Davis, of Nashua, and Manager L. F. Carter, of the Metropolitan Casualty Company, presented a plan for health and accident insurance for the members of the society. A motion was made and passed to accept this insurance plan and to authorize the committee of five to make the necessary arrangements.

On April 2, 1941, a noon meeting was held at the Eagle Hotel; about twenty-five were present. The Orthogynol Company presented a motion-picture film with sound track, "Studies in Human Fertility: Methods for the control of conception."

HENRY H. AMSDEN

ROCKINGHAM COUNTY

Rockingham County Medical Society has held two meetings during the past year, the spring meeting at the Portsmouth Hospital and the fall meeting at the Exeter Hospital.

At these meetings, cases were presented showing end results. At the fall meeting, a paper was read by Dr. Robert Zollinger, of Boston. At the spring meeting, Dr. Ezra Jones, of Manchester, talked on various difficulties in treating many types of fractures and Dr. A. Peters discussed the use of lipiodol in determining the patency of the fallopian tubes by x-ray. The meetings were very interesting, instructive and well attended.

HERBERT L. TAYLOR

STRAFFORD COUNTY

The Strafford County Medical Society has twenty-nine active members and six affiliate members. Two applications for membership were acted on at the April 30, 1941, meeting.

The annual meeting was held on October 30, 1940, at the City Hotel in Rochester. It was the one hundred and thirty-third meeting of this society and was attended by twenty-five members and one guest. Dr. Deering Smith, chairman of the State Committee on National Defense, spoke on the American Medical Association's plan to register all physicians in the United States and on their place in the national defense program. During the winter and early spring months, every physician in Strafford County was registered in accordance with this program.

Our spring meeting was held on April 30, 1941, at Simpson's Pavilion in Dover, with twenty-two members

and two guests present. Dr. Chester S. Keefer, of Boston, spoke, his subject being "The Modern Treatment of Pneumonia." Considerable business was transacted, which included: voting two new Rochester physicians to membership; voting Dr. A. P. Richmond, formerly of Dover, to honorary membership; appointing a committee to study the so-called "Houston Plan"; a vote against any change in the present Cancer Commission, and telegrams of such action sent to the secretary of the New Hampshire Medical Society and to Winslow Osborne, chairman of the Committee on Revision of Statutes, State House, Concord; presentation of Group Health and Accident Insurance for Physicians, which was favorably accepted. This was the most active meeting we have had for some time.

During the year 1940 one member was lost by death — Dr. L. L. Gilman, of Rochester, who died on April 12, 1940, at the age of seventy-one. Dr. Andrew J. Oberlander, formerly at New Hampshire University, accepted a position with an insurance company and moved to Burlington, Vermont. Dr. L. H. Mendelson, formerly of Dover, removed to Winchester. Dr. Dean Wilder, Dr. Oberlander's successor at New Hampshire University, went to Derry to become a general practitioner.

Dues of all the members were paid promptly and disbursed to the state society treasurer on February 8, 1941.

JOHN A. HUNTER

SULLIVAN COUNTY

The Sullivan County Medical Society held two meetings during the past year. The summer meeting was held at Seven Hearths, in Sunapee, and consisted of a luncheon followed by a scientific session, at which Dr. Ralph E. Miller, of Hanover, talked on laboratory technic and the need of laboratory examinations in general practice, particularly stressing frequent blood-sugar determination in the treatment of diabetes.

The winter meeting was held at Claremont. At this time officers were elected for 1941. The speaker was Dr. George S. Amsden, of Alstead, who gave a very interesting and instructive talk on the neurologic patient. We had hoped to have our state society president at this meeting and were much disappointed that he could not attend.

We are pleased to report the Sullivan County Society in a very healthy condition.

EMERY M. FITCH

Dr. D. G. Smith then made his report as delegate to the American Medical Association.

Report of the Delegate to the American Medical Association

The ninety-first annual session of the American Medical Association, held in New York City, June 10–14, 1940, was the best attended medical meeting that has ever been held, the total registration being 12,864, more than 2500 greater than any previous session. Thirty-nine New Hampshire doctors were registered, and your delegate served as sergeant-at-arms at the meetings of the House of Delegates. The secretary reported the membership of the American Medical Association to be 116,266, an increase of 15,000 members in five years, which is proof of the solidarity of the medical profession. Dr. Rock Sleyster, the president, remarked on how little the average doctor knows about the association and what it is doing. He recommended the training of speakers who could appear on the programs of the various county societies, to bring home to the doctors the activities and policies of the American Medical Association.

The House of Delegates recommended "to all state medical societies that they hold conferences with the authorities of their state boards of health, with the view of limiting the type and extent of services offered by the laboratories of these organizations. Laboratory services by the state board-of-health laboratories should be confined to requests made by health officers and to those made by physicians whose patients find it difficult or impossible to pay the cost of laboratory services of this kind in the customary manner. In general, laboratories of the state boards of health should not provide services at taxpayers' expense to persons who are able to provide for themselves."

The by-laws were amended by creating a new section of the scientific assembly, the Section on Anesthesiology. It was also proposed to amend the constitution so that only doctors of medicine licensed to practice medicine and whose licenses are registered in the state or county in which they make application be accepted for full membership in the American Medical Association. The determination on the part of the Board of Trustees to use every effort and means to defend the American Medical Association against the charges that it is guilty of conspiracy and of violating antitrust laws received the wholehearted endorsement of the House of Delegates. New England was honored by the choice of Frank H. Lahey, of Boston, as president-elect.

Medical preparedness was the major topic discussed at the meetings of the House of Delegates. Colonel George C. Dunham submitted a tentative plan for the procurement of professional personnel for the Medical Corps of the United States Army in the event of a national emergency. This plan was prepared by the office of the Surgeon General of the United States Army and was endorsed in principle by the House of Delegates. The House of Delegates created the Committee on Medical Preparedness, which is to "establish and maintain contact and suitable relationship with all governmental agencies concerned with the prevention of disease and the care of the sick, in both civil and military aspects, so as to make available at the earliest possible moment every facility that the American Medical Association can offer for the health and safety of American people and the maintenance of American democracy."

The president and secretary of the New Hampshire Medical Society were asked to recommend a chairman for this state. Following their recommendation, Dr. Deering G. Smith was appointed New Hampshire chairman by the Committee on Medical Preparedness of the American Medical Association. Dr. Smith chose Dr. Ezra A. Jones and Dr. Carleton R. Metcalf to serve with him on the state committee. On the suggestion of the state chairman, the president and secretary of each of the county societies appointed medical preparedness committees in their counties.

At the request of the Surgeons General of the United States Army, Navy and Public Health Service, a questionnaire was sent to every licensed physician in the United States in order to have an inventory of the doctors and their capabilities. The county medical preparedness committees have co-operated in getting doctors to complete these questionnaires, and blanks have been returned for every doctor in this state. The information on these questionnaires has been copied onto punch cards so that it is possible by the use of the usual machines to pick out any special group of physicians.

The carrying out of the Selective Training and Service Act of 1940 necessitated the use of physicians as examining physicians of the draft boards and as members of the medical advisory boards. The president and secretary of the Society made recommendations of doctors to the Governor, who nominated the physicians for the President to appoint, and who appointed the members of the medical advisory boards.

It was found necessary to use some civilian physicians for the final examinations for the United States Army. Volunteers were asked to serve on the medical induction boards, and over thirty physicians answered this call.

Dr. Smith spoke on medical preparedness at a majority of the county medical meetings in the fall, has attended several conferences, and has directed this work in the State. Every effort is being made to provide for the medical care of the United States Army and at the same time to look after the needs of civilian hospitals and of cities and towns, especially to prevent the stripping of rural and isolated communities of their necessary medical personnel. Information has been sent to the Corps Area Surgeon as to which medical reserve officers may be spared for active duty at this time and which officers should remain at home to take care of the needs of the community or its hospitals. At the present time each county medical preparedness committee is making a similar report about the other physicians in its county. The names of physicians who are deemed essential for the medical staffs of hospitals and related institutions, health departments and medical schools are being listed, and also the names of physicians who are needed for full-time industrial practice and for the care of the civilian population.

Eight New Hampshire doctors are now in active federal service with the National Guard, and at least sixteen other doctors are now serving a year's active duty as medical reserve officers. The practices of these physicians and of physicians who will subsequently go into the military service should be protected, and the House of Delegates recommended to the constituent state and component county societies that they work out plans whereby the practice of physicians absent from their homes in the service of our military and naval forces be protected, and whereby some financial return from such practice be secured for the physicians so absent in the service. Your delegate recommends that this matter be referred for study and action to the Committee on Medical Economics.

It is suggested that a committee on medical preparedness be chosen by this society, so that no question of authority may arise when we are dealing with the state and federal governments.

Your delegate appreciates the excellent work that the examining physicians of the draft boards and the members of the medical advisory boards have been doing. He has had the full co-operation of all the doctors of the State, and especially thanks the members of the medical preparedness committees for their assistance.

DEERING G. SMITH

As chairman of the Committee on Officers' Reports, Dr. D. G. Smith recommended that the part of his report dealing with medical defense should be read at the general meeting of the Society; it was so voted. After some discussion, the recommendation that the Committee on Public Health be instructed to study the problem relative to the State Laboratory as a real competitor of the clinical pathologists was laid on the table indefinitely. Dr. Smith's recommendation that the matter of protection for the practices of members who left the State because of military or naval service be referred to the Committee on Medical Economics for study and action was approved, as was the suggestion that a committee on medical preparedness be created, the suggestion being made that the New Hampshire Committee on Medical Preparedness of the American Medical Association constitute this new committee of the Society.

Dr. Woodman then presented the report of the Committee on Amendments to the Constitution and By-Laws.

Report of the Committee on Amendments to the Constitution and By-Laws

The Committee on Amendments to the Constitution and By-Laws met on March 31, 1941, and compared our constitution and by-laws with those of the American Medical Association. Owing to the excellent work of Dr. H. O. Smith and his committee, we find no discrepancies. We make the following recommendations:

1. It is recommended that, in Chapter VIII, Section 1, of the by-laws, the Nominating Committee be stricken from the list of standing committees. This committee is a special committee appointed for a special purpose and is provided for in Chapter V, Section 2, of the by-laws.

2. It is recommended that, in view of the above, Chapter VIII, Section 2, of the by-laws be stricken out entirely.

3. It is recommended that the list of standing committees shall include a committee on child welfare and maternal health.

4. It is recommended that a section be added to Chapter VIII defining the Committee on Child Welfare and Maternal Health as a committee consisting of three members whose duties it shall be to investigate the condition of child welfare and maternal health throughout the State, to report to the Society the cause of maternal and infant mortality and morbidity and to recommend any course of treatment or method of procedure that would improve such existing conditions.

5. It is recommended that each county society shall compare its constitution and by-laws with those of the state society and see that no conflict exists.

JAMES B. WOODMAN
CHESTER L. SMART
FREDERICK S. GRAY

Dr. D. G. Smith, acting for the Committee on Officers' Reports, approved the recommendations and proposed the following changes in the by-laws:

1. Chapter VIII, Section 1: strike out "A Committee on Nominations."

2. Chapter VIII: strike out Section 2.

3. Chapter VIII, Section 1: add "a Committee on Maternal Health and Infant Welfare" after "a Committee on Medical Education and Hospitals."

4. Chapter VIII: renumber Sections 3 to 12 inclusive, making Section 3, Section 2 and so forth.

5. Chapter VIII: add a new section, Section 12, as follows:

> The Committee on Maternal Health and Infant Welfare shall consist of three members whose duties it shall be to investigate the condition of maternal health and infant welfare throughout the State and to report to the Society the causes of maternal and infant mortality and morbidity and to recommend any course of treatment or method of procedure which would improve such existing conditions.

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The committee also recommended that, if the amendments were adopted, the Committee on Maternity and Infancy should be abolished. Since no action could be taken, the proposed changes were tabled until the next meeting. However, it was voted that it was the sense of the meeting for the Committee on Maternal Health and Infant Welfare and the Committee on Child Health to be standing rather than special committees.

Dr. Wilkins then presented the report of the Committee on the Control of Cancer.

Report of the Committee on the Control of Cancer

During the past year your committee has sent out three letters to every physician in New Hampshire. These one-page letters have dealt with three phases of cancer control.

The first was a quotation on cancer education for the physician, which was written by Dr. C. C. Little, the director of the American Society for the Control of Cancer. This letter dealt with the accomplishments in lay education that had been directed toward informing the public of the danger signals of cancer and an attack on fear of the disease. The unreasonable fear of cancer and discussion of it have been controlled to a considerable degree, and the education of the public has reached a point where there is definite need for further medical education, although this need varies in different states and communities.

The second letter dealt with the necessity of examining patients who have abnormal flowing even though the flowing is present at the time of the visit to the doctor. Too many patients have remained away too long a time when told to return when flowing ceased. Physicians were also urged to encourage women to have a vaginal examination between the ages of thirty-five and forty, and to advise cauterization and repair of cervical erosions when discovered. The importance of periodic examinations was also stressed in this letter.

In the last letter, yearly physical examination was stressed more fully, and certain diagnostic procedures were discussed that should be observed in making an examination. Your committee believes that if these rules were followed in all physical examinations, very few early cancers would be overlooked in the patients thus examined.

Here in New Hampshire we believe that the medical profession is constantly growing more alert in recognizing conditions that may indicate the possibility of cancer, and reports from the various diagnostic clinics in the State bear out this assumption, since more and more physicians are sending patients to the clinics for assistance in making definite diagnoses. The important educational work of the Women's Field Army continues, and through its efforts, hundreds of women have become interested in cancer control, and through their efforts thousands of pamphlets giving information in simple language have been distributed to homes throughout the State. These women as a group have many times praised the assistance and co-operation of the physicians in their communities.

Last July the Women's Field Army donated funds for the purchase of about six hundred copies of the book *Cancer Manual for Practitioners*. One of these volumes was sent to every member of the Society. We assume that every physician receiving this most informative book appreciated it, although the Women's Field Army had only ten letters of thanks.

New Hampshire is a small state, most of our physicians are intelligent and alert, and the New Hampshire Medical Society is a fairly compact group; hence your committee believes that there is no reason why the medical profession of this state should not lead other states in its ability to recognize cancer early and see that patients receive early and competent treatment. Your committee will gratefully receive any suggestions of further means and methods by which our educational efforts can be improved.

The committee has spent $42.57 for printing and postage, the balance of $7.43 having been returned to the Treasurer. We request another appropriation of $50 for the coming year.

GEORGE C. WILKINS

On the recommendation of Dr. D. G. Smith, of the Committee on Officers' Reports, the appropriation of $50 for the work of the cancer committee and the sending by the Secretary of a letter to the Women's Field Army expressing gratitude for the forwarding of copies of the cancer manual to all members of the Society were approved.

The report of the Committee on Medical Economics was read by the chairman, Dr. Robinson.

Report of the Committee on Medical Economics

The Committee on Medical Economics has continued the activities reported at the last annual meeting and presents the following report on the problems it has considered.

Medical Care for Clients of the Farm Security Administration. This program was in effect in Grafton and Cheshire counties for the full year of 1940. It appears to have worked out in general to the satisfaction of both the patients and the physicians concerned. All bills submitted by the participating physicians during the year were paid in full. Repayment of loans made to clients by the Farm Security Administration was in the neighborhood of 85 per cent, a fact indicating that, in the majority of instances, the program is fulfilling its original purpose of maintaining the self-sufficiency of the participating clients and has not degenerated into a government subsidy. The plan is being continued in Grafton and Cheshire counties for the year 1941. The prospects for successful operation are somewhat more uncertain this year, since shifting economic conditions are causing migration of labor from farm to industry, and the number of families participating will probably be smaller than that of last year. Your committee will continue its observation of the

operation of the program, and makes no specific recommendations at the present time.

National Physicians Committee for the Extension of Medical Service. This committee has done valuable and effective work during the past year in interpreting the aims and ideals of the medical profession to the public, and in combating undesirable trends toward government interference in the practice of medicine. Since it is a vital concern of physicians today that the public be educated to a sympathetic understanding of their position, and since the National Physicians Committee appears to be an effective and ethical agency for the attainment of this purpose, your committee recommends the continued moral and financial support of the committee, both by physicians individually and by the county and state societies. Specifically, it is recommended that the state and county societies make use of the news releases furnished by the National Physicians Committee for insertion in local newspapers.

Group Sickness Insurance. Your committee has continued its study of various plans now in effect for group insurance against the costs of medical care. It has considered the advisability of a fact-finding survey of conditions in New Hampshire, but has concluded that it would be useless to conduct such a survey at the present time, in the face of rapidly changing economic conditions. Furthermore, it is the opinion of your committee that with the expanding opportunities for employment that exist at present and will probably continue to exist for some time to come the need for group insurance against the costs of sickness is less acute than it has been in the recent past. Your committee believes, however, that the New Hampshire Medical Society should be prepared to introduce such a program if and when it should seem desirable in the future. Since a prerequisite of any such program would be the passing of an enabling act by the Legislature, it is recommended that the Committee on Public Relations or the Committee on Medical Economics be instructed to consult with Attorney Sulloway with the purpose of drawing up such a bill for later submission to the Legislature.

RICHARD W. ROBINSON, *Chairman*
CLARENCE E. DUNBAR
LESLIE K. SYCAMORE

Dr. D. G. Smith, for the Committee on Officers' Reports, expressed gratification that the plan for the medical care for clients of the Farm Security Administration was in successful operation, approved the work of the National Physicians Committee for the Extension of Medical Service, and urged that the news releases of the latter organization be used by the secretaries of all state and county medical societies. He recommended that the Committee on Public Relations and the Committee on Medical Economics consult with Mr. Sulloway relative to enabling legislation for group insurance against the costs of medical care and report back to the House of Delegates at its next annual meeting; after some discussion, the motion was passed.

The report of the Committee on Medical Education and Hospitals was then presented.

Report of the Committee on Medical Education and Hospitals

POSTGRADUATE MEDICAL EDUCATION

The New England Postgraduate Assembly will hold its fourth annual session in October on dates which will not be in conflict with any state medical society meeting or other major medical function. On the recommendation of your representative, the emphasis in the program will be from the viewpoint of the general practitioner.

A preliminary program will be issued this spring under the direction of the co-chairmen, Drs. Chester S. Keefer and Joseph H. Pratt, for medicine, and Drs. Howard M. Clute and William E. Ladd, for surgery. The success of the previous programs is a guarantee of the value of the next. An attendance of eight hundred, a modest expectation from the five states, will assure the necessary income for expenses, thus obviating underwriting by the Society. Each member should begin now to formulate plans permitting attendance.

At the fourth annual session of the Associated State Postgraduate Committees held in New York City on June 12, 1940, the new chairman, Dr. Frank R. Ober, of Boston, appointed a committee to study a proposed national registry of postgraduate instructors and meetings. The New England member is Dr. Frederick T. Hill, of Waterville, Maine. In the meantime an interstate mailing list has been established, permitting a nation-wide exchange of information, which is especially important in these days of constant scrutiny from without and within that call for continual alertness in how to provide the best grade of medical service for the protection of public health in the national interest, in which postgraduate instruction is a most significant factor.

COMMONWEALTH FUND FELLOWSHIPS

Postgraduate medical education in our own society has been significantly influenced in the past half decade by the fellowships provided by the Commonwealth Fund. It seems appropriate at this time to present a summary of these because of their importance as historical evidence of trends in the State, and because the bare numbers might appear to constitute something of a challenge to a certain age group in the Society when considered in the light of the fact that almost without exception the applicants who have been out of medical school five years and who have not been more than forty-five years of age have received awards within the limits of the fellowships available for any particular year.

It might be considered, on the other hand, from the basis of the same bare numbers that the Society has made a very respectable showing. It would be impossible to determine exactly how many fellowships under their specifications the directors of the Commonwealth Fund might have awarded during the period assuming an unlimited supply, but a statistical study discloses that somewhere between one fifth and one third of all those who might have been eligible did receive fellowships.

Sixty fellowships were awarded to thirty-seven of forty-two applicants; twenty-one fellowships were single, while ten awards were double. Five physicians received three grants, and one received four. Over half (thirty-three) were in medicine, ten in obstetrics, eight in pediatrics, seven in surgery, and two in x-ray. Two took medicine twice, and one each repeated pediatrics and x-ray.

In 1936 there were eight, in 1937 fourteen, in both 1938

and 1939 eleven, and in 1940 sixteen applications, in contrast with six for 1941. Each year new eligibility comes into existence and older practitioners are still considered on the basis of their individual qualifications. The directors of the Commonwealth Fund have shown a broad and most intelligent understanding of the problems of practice in the State, and it is to be hoped that our members will express their appreciation by continuing to take advantage of this opportunity of maintaining to an increasing degree a high level of professional competency.

JOHN P. BOWLER, *Chairman*
JAMES W. JAMESON
HERBERT L. TAYLOR

(To be continued)

MEDICAL PROGRESS

THE TREATMENT OF DEGENERATIVE JOINT DISEASE*

CHARLES L. SHORT, M.D.,† AND WALTER BAUER, M.D.‡

BOSTON

AS WILL be noted by the title, we prefer the term, "degenerative joint disease," to any of the more commonly used synonyms: hypertrophic arthritis, osteoarthritis or senescent arthritis. Our reasons for this choice are, first, because it is now generally agreed that degeneration of the articular cartilage is the primary pathologic change[1–15] and, secondly, because true arthritis, which suggests inflammation and even infection, is very rarely present.

Degenerative joint disease is without doubt the commonest articular disorder. All persons beyond the second decade of life, although they may not complain of skeletal symptoms, exhibit anatomic alterations in their joints similar to the pathologic changes observed in this disease. The agent responsible for this articular lesion has a scope of unusual width, as indicated by the facts that many animal species[4, 8, 12] in dissimilar geographic locations and persons of both sexes are affected, and that not only the articular coverings but also the hyaline cartilage in various sites of the organism participate in the characteristic changes. Although sequelae identical to degenerative joint disease may supervene on the subjection of articular cartilage to a great variety of pathologic influences, — such as trauma, disturbed joint mechanisms and inflammatory joint disease, — there remain a very large number of cases of this disease in which no such factor appears to have been operative. Regardless of etiology, however, two fundamental considerations apply to every example of degenerative joint disease. One is the disadvantageous biologic position of the articular cartilage when it meets with injury; this is evident, anatomically, in the paucity of its cellular constituents and in its remoteness from blood supply and, physiologically, in its low respiratory rate and in its extremely limited faculty of autogenous repair.[16] The other concerns the effect of mechanical stress and strain incident to joint function on a tissue whose specific means of resistance to such use has been depleted. It is this second group of forces that plays the dominant role in the localization and progression of the arthropathy to the late and conspicuously deforming changes. The most constantly observed correlative to the so-called "primary form" of degenerative joint disease is increasing age. This has been well demonstrated in a recent report.[15] From this study, it is apparent that further knowledge concerning the cause and prevention of this articular lesion depends on precise data concerning the factors that govern maturation, maintenance and senescence of hyaline cartilage and other mesenchymal tissue. The best chance of obtaining this much-needed information resides in the development of better staining technics, the combination of microscopic observations and histochemical methods, and studies in tissue metabolism.

Those who have the misfortune to acquire pain and disability as a result of their degenerative joint-disease changes can be divided into two groups. In the first group, which might be termed the "primary or endogenous type," a factor as yet unrecognized is at work. In the remainder, which we may call the "secondary or exogenous type," long-continued trauma is the agent that oftenest favors the development or acceleration of hyaline-cartilage degeneration. The various agents that may be operative and a knowledge of which is of value in reference to the

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*From the Medical Clinic, Massachusetts General Hospital, the Department of Medicine, Harvard Medical School, and the Massachusetts Department of Public Health.

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‡Associate professor in medicine, Harvard Medical School; physician, Massachusetts General Hospital.

treatment and to the prophylaxis of this condition are as follows: long-continued trauma, such as that in occupations demanding persistent use of one or more joints and that in obesity, in which the weight-bearing joints are subjected to unusual strain; static deformities resulting from severely pronated feet, knock-knees or bowlegs, poor posture (with obesity often a factor), fractures healing with malalignment, displaced patellas, incompletely healed sprains or unreduced dislocations or diseases of the epiphysis; and intra-articular disease, previous or present, which is the result of trauma (including osteochondritis dissecans, internal derangements of the knee joint, fractures into the joint and so forth), sepsis (joint infections due to the gonococcus, pyogenic organisms and so forth), gouty arthritis or rheumatoid arthritis.

It is thus apparent that degenerative joint disease may follow many types of articular injury and may often represent the end stage of other types of arthritis. Accordingly, the physician should not be satisfied with a clinical or roentgenologic diagnosis of this condition without first ascertaining whether one of the above-mentioned factors is of causative importance. Furthermore, he must make certain that the degenerative changes are actually responsible for the patient's symptoms, and that they are not merely a coincidental finding.

The onset of degenerative joint disease is usually gradual and the course chronic. Differentiation from the various acute and chronic arthritides is rarely difficult. As previously pointed out,[17] it is of extreme therapeutic and prognostic value to distinguish degenerative joint disease from rheumatoid arthritis. Patients showing evidence of both diseases should not be given the diagnosis of mixed arthritis. It is the physician's duty to decide whether the two diseases occupy a causative or a coincidental relation.

Since articular cartilage, because of its physiologic limitations, has a limited ability to repair itself, anatomic cure of degenerative joint disease is not possible. On the other hand, treatment directed toward the correction of causative or accelerating factors (if present and reversible) will arrest the progress of the disease and, in conjunction with simple measures to be described, will relieve the patient's symptoms.

Articular cartilage itself is insensitive. The pain in degenerative joint disease is therefore due to secondary changes, including muscle spasm caused by mechanical strain, loose bodies, secondary inflammation of fibrous tissue or bursae and elevation of the periosteum from the marginal overgrowth of cartilage and bone. The first three may be relieved by treatment, notably rest and removal of irritating factors, and the last tends to subside when proliferation of cartilage ceases and calcification takes place. The most easily observed example of this process is seen in degenerative disease of the terminal phalangeal joints (Heberden's nodes), which often begins as a tender painful swelling that, in the course of a few months, becomes ossified and relatively painless. This tendency to self-limitation must be recognized in the evaluation of any form of therapy and has undoubtedly led to false claims of success from methods that rest on neither a theoretical nor a clinical basis; these include removal of foci of infection, vaccines, dietary regulation (with the exception of reduction diets) and endocrine therapy. In spite of many claims to the contrary, critical evaluation of present evidence is entirely against an infectious, metabolic or endocrine etiology for degenerative joint disease.[11]

One of us (W. B.[17]) has stressed elsewhere the necessity for a careful distinction between degenerative joint disease and rheumatoid arthritis. Once the physician is sure of his ground, the first essential step is to explain the nature of the condition to the patient, thus dispelling the fear of crippling and disability that has undoubtedly arisen in the latter's mind and has been fostered by the comments of his associates. For this reason, it is better to avoid the use of the term, "arthritis," altogether, on account of its gloomy connotation and to stress the favorable prognosis possible for the preservation of function and the relief of pain.

In brief, the principles of treatment of degenerative joint disease consist in reassuring the patient and explaining to him the nature of the disease, correcting responsible factors, if present, and affording symptomatic relief, so far as possible. Since it is often impossible in a given case to separate symptomatic measures from those that have a more direct effect on the progress of the disease, no attempt will be made to do so in the following detailed consideration of treatment.

Methods of Treatment

Correction of Contributing Factors

Once the diagnosis of degenerative joint disease has been made and its nature and prognosis explained to the patient, the physician's next consideration should be to decide whether any irritating or accelerating factors can be removed or ameliorated. As a check, the previously listed contributing factors should be consulted. If occupational trauma is still going on, the patient may be advised to change or modify his job. Obesity obviously calls for weight reduction, which is dis-

cussed below. Foot strain and poor posture suggest the use of supporting plates, belts and corsets, and the strengthening of flabby muscles by exercise. The treatment of other static deformities usually requires orthopedic consultation. Intra-articular disease of a traumatic nature may demand surgery. If an infectious arthritis plays a causative role and is still active, specific treatment should be instituted at once.

Diet

None of the special diets that have been loosely applied to various forms of chronic arthritis are indicated in degenerative joint disease.[18] Protein, whether or not in the form of red meat, need not be cut down on account of the joint symptoms. Carbohydrates should be limited only if necessary to reduce weight. Whether or not the diet is "rough" depends on the patient's needs in regard to intestinal activity, diverticulosis and so forth. So-called "acid fruits and vegetables" bear no known relation to the degenerative process. From the nature of the pathogenesis of the disease, as described above, it is difficult to see how diet can play a direct role. Many claims for the successful use of certain diets undoubtedly depend on the spontaneous subsidence of periosteal irritation from encroaching marginal osteophytes. In short, the only known value of dietary regulation in degenerative joint disease lies in the field of weight reduction.

We have mentioned above that obesity may be a precipitating or accelerating factor in degenerative joint disease. Conversely, weight reduction will often relieve symptoms to a striking degree. In markedly obese patients, the indications are obvious, but relief is often gained by the loss of 10 to 20 pounds in patients only slightly overweight. It is of advantage to have the patient lose the first 10 or 20 pounds rather rapidly so that he may be encouraged to persevere from both the evidence of the scales and his less painful joints. The most striking benefit from weight reduction is in degenerative joint disease of the knees, which occurs in overweight women at or about the time of the menopause.

Climate

The symptoms of degenerative joint disease may frequently be brought out by exposure to dampness or cold. For this reason, it is wise to caution the patient against such exposure and to insist on warm clothing when necessary, including gloves for patients with Heberden's nodes and long underwear for those with knee involvement. If practicable for those living in northern latitudes, much discomfort and even exacerbations may be avoided by migration to a warm, dry climate during the winter season.

Drug Therapy

No specific drugs are known that will alter the course or permanently do away with the symptoms of degenerative joint disease. None of the recently introduced so-called "specifics" for the treatment of arthritis have proved efficacious when tested under controlled conditions; these include gold salts, sulfur and massive doses of vitamin D.

Analgesics are of real value, but should not be used to the avoidance of rest, correction of irritating factors and other measures of proved worth. Aspirin, in regular dosage of 10 gr. (0.6 gm.) every three or four hours, is effective and inexpensive. Five grains (0.3 gm.) of phenacetin or ¼ to ½ gr. (0.015 to 0.03 gm.) of codeine may be added to each dose of aspirin, if necessary. Compounds containing amidopyrine or cinchophen had best be avoided owing to the possibility of rarely occurring but serious toxicity. If morphine is needed, the physician should be suspicious that some other serious condition is also present, notably metastatic cancer.

Potassium iodide has long enjoyed a favorable reputation in the treatment of degenerative joint disease, but in our experience no benefit has been derived from the use of this drug that could not be ascribed to other measures or to the natural course of the disease.

The use of small doses of thyroid is permissible to increase metabolism and thus aid in combating obesity, but no specific effect has been proved from the use of this drug or other endocrine preparations. Of course, if myxedema is present in a patient with degenerative joint disease, the symptoms may be aggravated. In such cases, the use of thyroid is indicated. Similarly, if the patient is a woman with menopausal symptoms, replacement therapy with estrogenic material may be helpful, but no direct effect on the joint disease can be expected.

Rest

Hospitalization is rarely necessary for patients with degenerative joint disease. Diagnosis can usually be made in the office with the benefit of a careful anamnesis and examination, in addition to roentgenograms of representative joints and routine examination of the blood and urine, with the occasional aid of the blood sedimentation rate. In a few patients, symptoms due to traumatic synovitis with effusion are sufficiently acute to require hospital care. A few others, of course, require hospitalization because of necessary surgery. The majority of patients can benefit by

rest at home according to the directions of the physician, often without interfering with housewifely duties or means of livelihood. The prescription of rest should be definite and included in the written directions, which are of great advantage in the management of a patient with chronic joint disease. It is obvious that relief of strain to the weight-bearing joints, which are the usual sites of degenerative joint disease, can be gained by a sitting or preferably reclining position for certain intervals during the day. The amount of rest prescribed varies with the severity of symptoms and may be gauged by trial, the relief of pain being the criterion. A good start may often be made by getting the patient off his feet for a week or two, followed by rest periods for one or two hours during the day. If work must be carried out, the patient can rest in the noon hour and at home before the evening meal. Finally, the amount of rest prescribed must be tempered by two considerations. In the first place, the patient must not believe that he is a permanently crippled invalid, cut off from his usual occupations and hobbies. At every point, the rationale of treatment must be explained, and the relatively good prognosis emphasized. A second consideration is that, in common with other forms of articular disease, the joints tend to become stiff and painful if kept in one position too long.

In addition to rest for the patient as a whole, the involved joints may acquire protection from strain by the use of certain advantageous positions or mechanical supports. For the cervical spine, periods of rest in a supine position, with no pillow or a low one, are of benefit. For severer symptoms, a Thomas collar or head traction is necessary. Similarly, for involvement of the lumbar and sacroiliac articulations, the patient should lie on the back with the knees and hips flexed during rest periods. A hard mattress should be employed, and boards may be placed between mattress and spring. When the patient is up and about, strain to the spinal and sacroiliac joints may be avoided by a corset—a brace is rarely necessary. If the patient drives a car, he should be able to reach the pedals readily with the knees flexed; if necessary the seat should be moved forward or a pillow should be placed behind his back. Painful hips may be relieved for a time by rest in bed, with traction on the leg or the use of a spica of plaster or leather. Elastic bandages or, better, a laced, part-leather, part-elastic kneecap takes from the knees much of the stress and strain of walking. Crutches allow the patient to be ambulatory, yet take the strain off a painful hip or knee. If the feet are involved, usually only in the secondary type of degenerative joint disease, support and correction of faulty mechanics can be gained by the use of heel lifts and footplates. The aid of the orthopedist can be sought with advantage in planning these measures for the care of individually involved joints.

Physical Therapy

Symptomatic relief, one of the chief aims of therapy in degenerative joint disease, can be aided by physical therapy in the form of heat, massage and graded exercises. The mechanism of relief by the use of heat is thought to be increased blood flow, although there is no evidence that the circulation to joint tissue is impaired in this disease. Simple methods of providing heat to the joints are perfectly satisfactory and have the added advantage that they can be applied several times daily in the home. These methods include the use of hot packs, hot baths and an inexpensive electric-lamp baker. Diathermy and other electric machines using short-wave modalities for heating the deeper tissues are no more effective than the simple methods. The physician should give definite directions to the patient concerning the frequency and duration of treatments. In general, heat may be applied for periods of not over fifteen minutes, two or three times daily. A state of chronic passive congestion and tissue hypersensitivity can be induced by overenthusiastic baking. Gentle kneading or light stroking massage of the muscles surrounding affected joints can be applied following the administration of heat. Heavy massage or massage over the joint itself provides a harmful form of trauma, which may increase the patient's symptoms. It is therefore essential that the physician give exact directions for this method of treatment, whether it is administered by a member of the family or a trained physical therapist.

Exercise

Exercise, like rest, can be divided into that of the patient as a whole and that of the involved joints. In general, although a moderate amount may be necessary for weight reduction, exercise should be prescribed with caution. The patient should be informed that in this disease of the joints, activity is not necessary to prevent ankylosis, and overexercise may bear a definite possibility of harm. Frequently, pursuit of the patient's usual occupation will be all that the joints can stand. Violent sports should be avoided, especially those involving maximal use of an affected joint. Postural exercises, which are important to relieve weight-bearing joints of unnecessary strain, should be increased gradually—never beyond the boundary of pain. The exercise program for

strengthening the muscles that control the affected joints is much more important than the use of heat or massage. So long as the joint is inflamed and the muscles that control it are fatigued, they should receive complete rest. When spasm and inflammation have subsided, carefully graded muscle-setting exercises are begun and are persisted in daily until satisfactory power has been regained.

Psychotherapy

We have already alluded to the value of reassuring the patient and explaining to him the nature of degenerative joint disease. If freed from fear of crippling, the patient often bears with equanimity the occasional aches and pains that treatment fails to relieve. Other patients, especially those complaining of undue fatigability, have a lowered threshold of sensibility, which accentuates symptoms that might be borne without complaint by more stoical persons. In such cases, the lessening of domestic or business cares, vacations with change of scene, and the search for and eradication of disturbing elements in the environment are all-important in securing a satisfactory therapeutic result. For these patients, regular doses of a mild sedative, such as ¼ gr. (0.015 gm.) phenobarbital after meals and at bedtime, may be more helpful than the use of analgesics.

Special Types of Degenerative Joint Disease

Heberden's nodes. Degenerative joint disease of the terminal joints of the fingers is probably the commonest type seen in general practice. Rheumatoid arthritis can usually be readily distinguished, even without the aid of x-ray study and sedimentation rate; one should bear in mind, of course, that the two diseases may be present in the same patient in an unrelated form. It must also be remembered that the process may spread to the proximal joints in 10 to 20 per cent of the cases, or even rarely begin there. Acute cases, with marked tenderness, pain and occasionally fluctuation, must not be confused with acute gouty arthritis or sepsis. This condition is commoner in women than in men, and usually appears around the age of fifty. Younger patients with Heberden's nodes often give a history of premature development of the nodes in other members of the family. There is no specific treatment for this benign form of degenerative joint disease. The patient is more comfortable if the hands are kept warm and if trauma to them is avoided. The important thing is to explain the situation to the patient, who usually fears the worst, and assure him that he is not going to develop a crippling disease and that the nodes, if acute, will subside in the course of a few months, whether treated or not.

Temporomandibular involvement. As recently described by Costen,[19] a variety of symptoms may result from degenerative disease of the temporomandibular joint, caused by loss of molar support or by malocclusion. These complaints include otologic symptoms (deafness, tinnitus, pain, dizziness and nystagmus), headache (occipital and vertical), herpes of the external auditory canal and buccal mucosa, and burning or dryness of the mouth and tongue. Roentgenograms may show loss of substance of the head of the condyle anteriorly and of the articular eminence. The insertion of cork disks between the molars may give temporary relief and may serve as a therapeutic test. Permanent relief of symptoms belongs to the sphere of dentistry.

Morbus coxae senilis. The treatment of degenerative disease of the hip joint, morbus coxae senilis, calls for an individually designed program for each case.[20] If there is flexion-adduction deformity, a period of complete bed rest, with adhesive traction of the leg, may be necessary. Heat and massage, as described above, should be given daily. When the muscle spasm has subsided, carefully graded exercises for the hip abductors and extensors (the gluteus medius and maximus muscles) should be prescribed and followed over a prolonged period. Four to ten slow voluntary contractions or so-called "sets" of each muscle group at one sitting are repeated two or three times daily unless there is evidence of strain or fatigue. Unilateral hip disease may be protected from the strain of weight bearing by crutches. In some cases, restriction of motion by means of a leather or elastic knitted hip spica gives great relief. If there is apparent shortening of the leg due to fixed adduction contracture, the heel of the shoe should be raised sufficiently to compensate for the apparent inequality of leg length.

This is the one form of degenerative joint disease that may lead to actual crippling in spite of the conservative measures described above. Orthopedic consultation is in order for such cases to determine the advisability and type of operation. In an occasional patient, re-establishment of proper weight-bearing lines and improvement of function may be gained by osteotomy of the femur. In severe cases, help can be given by a plastic operation on the anterior wall of the acetabulum[21] or, better, by means of a vitallium-cup arthroplasty.[22] In the hands of the originator,[21, 22] these operations have been extremely successful in relieving pain and increasing the range of joint motion in

severe, incapacitating cases. Such an operation can be applied equally well to young patients with degenerative changes secondary to a slipping femoral epiphysis, Legg–Perthes disease and intrapelvic protrusion of the acetabulum.

Surgery

There are few indications for surgery in the treatment of degenerative joint disease, except for those already mentioned. One is the removal of loose bodies or so-called "mice" from the knee joint if pain, locking or secondary inflammation is caused thereby. Conservative treatment should be given a trial, but if incapacitating symptoms persist and the loose bodies are demonstrable by examination or roentgenograms, removal should be carried out without delay, since the degenerative process may be hastened by their presence.

Acute traumatic effusions usually subside with rest and splinting; if they persist, aspiration should be done.

Prophylaxis

Measures to prevent the development of degenerative joint disease or the acceleration of its course, once present, should be obvious to the reader on consulting the list of contributing factors already given. Such measures fall properly into the field of preventive medicine, and constitute one of the responsibilities of both family physician and specialist. In handling fractures and sprains, diseases of the epiphysis, rickets, infantile paralysis and so forth, the physician must bear in mind the possibility of premature development of degenerative joint disease if the joints are left subject to unusual strain. Static deformities and postural defects should be corrected as speedily and as completely as possible. Obesity, especially in those approaching middle age, is hazardous to the well-being of the joints, as well as of the other bodily systems. Repeated joint trauma, whether occupational or recreational, should be advised against. Finally, in a patient with any form of arthritis, the possibility of secondary degenerative changes must always be kept in mind and guarded against, especially if the movements of weight-bearing joints tend to become limited in a position of mechanical strain.

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CASE RECORDS OF THE MASSACHUSETTS GENERAL HOSPITAL

ANTE-MORTEM AND POST-MORTEM RECORDS AS USED IN WEEKLY CLINICOPATHOLOGICAL EXERCISES

FOUNDED BY RICHARD C. CABOT

TRACY B. MALLORY, M.D., *Editor*

CASE 27301

PRESENTATION OF CASE

A thirty-seven-year-old housewife entered the hospital with the complaint of joint pains.

About eleven years before entry, the patient began having mild attacks of pain in the shoulders and hands, which recurred every spring and lasted about two weeks. These attacks were relieved by salicylates and applications of heat. Three years later, her tonsils were removed for this condition. The following year, she developed a painful swollen ankle, and the condition soon spread to most of her other joints. Since then, she had had intermittent joint pains varying in intensity from time to time and usually aggravated by cold weather. The pain was not severe enough to keep her from doing her housework. Three years before entry, she developed a cough, with substernal pain and fever but no chills. She entered another hospital, where she was found to have bilateral pleural effusions. The pleural cavities were tapped twelve to fifteen times, and she developed empyema on the right, which was drained for several weeks. For the next three years, the joint pains were severer, but the patient was able to do her housework. For a year before entry, she had slight dyspnea on exertion and edema of the ankles, which disappeared at night. Two months before entry, she had a sudden onset of nausea, vomiting and diarrhea, with some pain around the umbilicus. The pain was somewhat relieved by vomiting but made worse by defecation. She had twelve to fifteen bowel movements a day, which were said to contain mucus and blood. The diarrhea ceased after treatment with Fuller's earth, and the patient improved considerably. A few days later, six weeks before entry, she developed severe dysphagia, at first with solid foods and finally with liquids; she was unable to swallow anything because of the severe pain and vomited about once a day.

She re-entered the outside hospital, where she was found to be obviously ill, pale and somewhat emaciated. Her left chest was dull to flat posteriorly below the angle of the scapula, and distant bronchial breathing and egophony could be heard in this area. The heart was moderately enlarged to the left, the rate was 120 and grossly irregular, and the sounds were of fair quality. No murmurs were heard. The blood pressure was 110 systolic, 60 diastolic. The abdomen was normal. An x-ray film of the chest showed fluid at the left base; the heart was displaced to the left. The blood showed a red-cell count of 3,470,000 with a hemoglobin of 65 per cent, and a white-cell count of 4000 with a normal smear. The blood Hinton reaction was negative, as were Widal and undulant-fever agglutination tests. A gastrointestinal series was negative except for "slight delay with slight narrowing at the extreme lower end of the esophagus, consistent with a mild degree of cardiospasm." A nasal tube was inserted. The patient was given fluid through the tube for forty-eight hours, after which she was able to take fluids and soft solids by mouth. The irregular heart beat proved to be paroxysmal, the attacks lasting a few hours. Her temperature and respirations were normal. After ten weeks, she was discharged on a bland diet. One month later, she entered this hospital for study.

Physical examination revealed a somewhat undernourished woman with signs of fluid or of a high diaphragm on the left, and a heart enlarged to the left, with sounds of good quality and no murmurs. The abdomen was normal. There was slight to moderate pain on motion of the neck, back, elbows, shoulders and knees. There was swelling and tenderness of a few of the small joints of the hands.

The temperature was 98.6°F., the pulse 90, and the respirations 20.

Examination of the urine was negative. The blood showed a red-cell count of 3,780,000 with 65 per cent hemoglobin, and a white-cell count of 4300 with 86 per cent polymorphonuclears. The nonprotein nitrogen of the blood serum was 19 mg. and the fasting uric acid 4.7 mg. per 100 cc.; the sedimentation rate was 1.94 mm. per minute. The blood Hinton reaction was negative.

X-ray study showed moderate decalcification of the lumbar spine and hands. There was slight narrowing of the vertebral joints, and some surrounding soft-tissue thickening. The chest showed enlargement of the heart to the left, with a rather prominent pulmonary conus. There was marked thickening of the pleura along the right axillary line from the apex to the diaphragm, and a small collection of fluid at the left base partially obscured the diaphragm and the apex of the heart. There were small areas of calcification at both lung roots. The nasal sinuses were clear. An electrocardiogram showed low T_1 and T_2, inverted T_3, shallow T_4 and normal Q_4.

The patient was given a transfusion and discharged somewhat improved.

Two and a half months after discharge, she developed an ulcer on the left external malleolus, which did not heal. She also developed over the spine several flat nodules that were the size of a ten-cent piece and lasted for several weeks. Eight months after discharge, she suddenly had a severe shaking chill, with pain in the right chest, which was aggravated by inspiration. She became nauseated and vomited, and had a headache, but no stiff neck or increase in joint pains. She developed a temperature of 104°F., the blood pressure dropped, and the pulse became relatively slow. She re-entered the outside hospital, where physical examination revealed an obviously very ill woman with slightly cyanotic lips and fingernails. The heart was enlarged to the left, the sounds were loud, and there was a systolic murmur at the apex, with a suggestion of a gallop rhythm over the mitral area. The blood pressure was 85 systolic, 50 diastolic. Percussion of the chest was normal, but in the right mid-back there were crackling rales and definite bronchial breathing. The abdomen was slightly distended, and the liver edge was palpable 5 cm. below the rib margin and quite tender. There was a small ulcer on the external malleolus surrounded by an area of erythema.

The temperature was 105°F, the pulse 100, and the respirations 20.

The urine contained the slightest possible trace of albumin and 10 to 12 white blood cells per high-power field and occasional granular casts in the sediment. The blood showed a red-cell count of 4,100,000 and a white-cell count of 22,000, with 86 per cent polymorphonuclears. The sputum contained pneumococci, but the blood culture showed no growth.

Two days after entry, the area of rales and bronchial breathing had increased in size, and x-ray examination showed an area of consolidation at the right lung root. The blood pressure had risen to 126 systolic, 80 diastolic. The following day, the patient was symptomatically somewhat improved, but her temperature remained elevated and definite dullness could be detected between the angle of the scapula and the spine on the right. There was apparently a small amount of fluid in the abdomen, and there was slight edema over the sacrum and about the hips. Her urinary output became markedly decreased, and intravenous Salyrgan had no apparent effect. On the eighth hospital day, the urine contained a large trace of albumin, with occasional red cells and 10 to 15 white cells per high-power field, but no casts. The nonprotein nitrogen of the blood serum was 70 mg. per 100 cc., and the chloride was 88 milliequiv. per liter. The patient was given 1000 cc. of concentrated glucose and sodium chloride, but her urinary output remained low. Twitching of the muscles developed, and the breath became uriniferous. The edema of the back and abdomen increased, and numerous nodules, 0.5 to 1.0 cm. in diameter, could be felt in both supraclavicular areas. The temperature remained high, and the pulse and respirations became more rapid. On the tenth hospital day, her condition became definitely worse. She developed generalized convulsions and died. The total urinary output in the last four days was about 300 cc.

Differential Diagnosis

Dr. Alfred Kranes: It is difficult to know how to begin on this problem because there seem to be so many apparently unrelated and independent episodes. I think the simplest way is to discuss each one independently and try to fit them together, if possible, into some sort of coherent picture.

The illness apparently began when the patient was twenty-six, and joint pains occurred about once a year. The description of the pain at the onset is not diagnostic of any type of joint disease with which I am familiar, although as the disease progresses, rheumatoid arthritis seems to be more of a possibility than any other type of joint disease; however, one does not expect patients with that disease to have a recurrence lasting only two weeks and disappearing, leaving her perfectly well for the rest of the year.

Rheumatic fever is another unlikely possibility. The only type of joint disease that will recur and subside, permitting a patient to be well in between attacks, is gout, but there is very little in this story to make one consider that diagnosis very seriously. So that I think we shall have to leave unsettled the diagnosis of her joint disease for the time being and go on to the next episode, which is the development of bilateral pleural effusion three years before admission. One would like to know what type of fluid was removed from this patient's chest. That we are not told, since it was done elsewhere. I presume it was a serous effusion. One would also like to know over how long a period she was ill with these effusions, since she had twelve to fifteen taps. I should interpret the empyema as probably being the result of repeated taps and not part of the underlying disease. The effusions must have lasted at least several months to have been tapped as many times as recorded here. What produced the pleurisy is far from clear. The usual causes of pleural effusion do not seem to fit this picture. Tuberculosis, of

course, is the first thing one considers, but it is quite unusual to get bilateral tuberculous effusions. They do occur but not very commonly. Furthermore, it is extremely unusual for a tuberculous effusion to require tapping so often as this. Apparently, the fluid reaccumulated rapidly. I think the chief argument against tuberculosis is that the empyema cleared up in several weeks. I certainly do not believe a tuberculous empyema would clear up so rapidly. Other causes for effusion, such as cancer, do not seem to fit this picture very well. It would be unusual to have a bilateral effusion with a malignant neoplasm. The patient might have had lymphoma, but I think it is extremely unlikely. There seem to have been no signs or symptoms of cardiac failure or renal or liver disease. I shall therefore leave the question of the pleurisy unexplained for the while.

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The patient then developed a totally new chain of symptoms — nausea, vomiting and bloody diarrhea. No details are given, and I doubt whether any satisfactory explanation for it will be found, since it did not recur. The chances are that whatever produced it had subsided at the time of death, and very likely no anatomic explanation may be forthcoming for these symptoms. The patient also developed severe dysphagia, which may also have been related to the lesion causing the previous abdominal symptoms. We can speculate about the presence of some ulcerative lesion in the mucous membranes of the gastrointestinal tract, but there is no direct evidence. The symptom cleared up, and nothing further is said about it.

The patient again entered the hospital with a left pleural effusion, which apparently was asymptomatic. No fluid was present on the right, since the right pleural cavity was probably obliterated by the previous empyema. This time, her heart was found to be enlarged, and she was apparently having paroxysmal fibrillation, although the exact nature of the arrhythmia is not described. The x-ray report disturbs me a bit, because it states that the heart was displaced to the left. If that is so, one must assume that there was collapse of a considerable part of the left lung. There is no apparent reason for it, and I wonder if that is a correct observation. Whether the heart was displaced to the left, we shall have to leave open to question; I rather doubt it.

The physical examination when the patient entered this hospital is more or less confirmatory of what was previously found, except that there appeared to be less fluid than was described before. Whether that was removed in the meantime or absorbed spontaneously, we are not told. Again, the heart was found to be enlarged, and the rest of the physical examination was normal except for pain and swelling of the joints. The urine examination was negative, which is important since the patient died of renal failure; this is the only report we have of a urine examination during the whole illness. The secondary anemia and leukopenia are again confirmed, but the rest of the laboratory data are of no value except for an elevated sedimentation rate.

The electrocardiogram does not mean much to me except that the patient may have had some myocardial damage. Following her discharge, she developed an ulcer over the left malleolus, which persisted until her death some six or eight months later. There seems to be no adequate explanation for it. Nothing is said about varicose veins, and she apparently was not syphilitic. The nodules over her spine interest me, and I should like to know whether they were subcutaneous or in the skin, and whether or not the skin was discolored. The description here does not help one very much. The nodules disappeared, apparently, in several weeks. Then the terminal event occurred, which was quite obviously a pneumonia. The rest of the story is that of a patient dying in uremia. What the cause of this terminal renal failure is, one cannot say from the record. The first question that comes to mind in a patient with pneumonia is whether chemotherapy was employed. Was she given any sulfapyridine or sulfathiazole?

DR. CHARLES L. SHORT: No.

DR. KRANES: That is too bad; because I had a ready explanation for the terminal renal failure. We shall therefore have to look for other explanations. Patients with overwhelming acute infections occasionally die in uremia, as some postoperative patients do. The blood pressure drops, and the renal blood flow diminishes and is followed by oliguria, anuria and consequent uremia. That is possibly the explanation here. In addition, there may be some underlying diffuse disease of the kidney, which shut down terminally. It does not seem, however, like an ordinary acute glomerulonephritis.

The whole problem seems to resolve itself into whether this patient had a number of different diseases explaining these separate episodes, or whether one can tie them together under one diagnosis. It would be more logical to explain everything on one diagnosis in this particular case, because it seems to me that there are no satisfactory multiple diagnoses that could be made explaining these separate episodes individually. One would like to find a disease that would explain them all. It would have to be quite a widespread dis-

ease involving the small blood vessels of the body or some diffuse vascular lesion of which these episodes were local manifestations. So far as I can see, there are only two diseases that would adequately explain this picture. One is periarteritis nodosa. The other is disseminated lupus erythematosus. So far as I know the pains of periarteritis are much more likely to be muscular or neuritic, rather than articular, nor does the disease cause recurrent pleural effusions — I have never heard of its doing so, at any rate. Furthermore the laboratory evidence is against it. Patients with periarteritis nodosa usually have a leukocytosis and very often an eosinophilia, neither one of which was present in this particular case.

The diagnosis of disseminated lupus — "acute disseminated lupus" is a very poor term because one cannot readily speak of an acute disease that has apparently lasted eleven years — seems to fit this picture better than anything else. It explains to me practically everything this patient had. Joint pains are very common in that disease. Recurrent pleural effusions or involvement of any of the serous surfaces is an outstanding characteristic. The gastrointestinal symptoms can be explained on that basis because patients with disseminated lupus develop lesions of the mucous membranes. To be sure, the lesions are more likely to be in the mouth or pharynx, but they can occur in the lower gastrointestinal tract as well. It could also explain the cardiac symptoms, because it frequently involves the heart, especially the pericardium. We have no evidence of pericarditis here, however. Occasionally it involves the endocardium, producing atypical vegetations on the heart valves, and cases have been described in which the myocardium has been involved, although the pathology of the disease is not at all clear. The diagnosis of disseminated lupus would also explain the ulcer of the leg, since it occasionally produces ulcerative lesions on the skin. Finally, many of these patients die of uremia. It is well known that people with disseminated lupus may have very extensive renal involvement. It is also interesting to note that patients with this disease, very often up until the time of death, show no evidence of renal disease, and then with the advent of some acute infection develop uremia. On the whole, disseminated lupus explains the picture better than anything else.

But there are objections to it. The first is its duration. Rarely do people with disseminated or visceral lupus live this long. The second and more important objection is the fact that throughout the disease no mention is made of skin lesions, unless possibly the nodules described represented involvement of the skin. One hesitates to make a diagnosis of lupus without skin lesions, but in the disseminated type, the cutaneous manifestations of the disease are the least important part. Since everything else fits so well, I cannot allow the absence of skin lesions to deter me from making that diagnosis, which has one other great advantage, namely, that there are no characteristic pathologic lesions. I think among pathologists there is quite a controversy about whether or not there are any characteristic lesions. The consensus seems to be that there are not. That being so, Dr. Mallory may find very little aside from the acute terminal pneumonia. Consequently, although we may not be able to prove the diagnosis, Dr. Mallory will not be able to disprove it.

Dr. Short: I saw the patient on both admissions to this hospital, and four or five days before she died. At the time she was in this hospital, there were two diagnoses that we considered: one was Dr. Kranes's diagnosis of disseminated lupus, and the other was a form of rheumatoid arthritis, with pleural effusion, pericarditis and myocarditis. We had the same objections to lupus that Dr. Kranes had — the lack of any characteristic skin lesions. It is fair to say that she did have some small scattered papulopustular lesions, but we were never quite sure whether they meant anything or were just what one might see on anybody. The patient also had what seemed to be a mild acne rosacea; at least we thought it was, and did not consider it characteristic of lupus. The other objection was the duration of the disease — she had been ill for ten years, although she was in relatively good shape. When I saw her just before she died, I could add nothing more to a consideration of the primary diagnosis. I thought she had an intercurrent pneumonia and cardiac failure.

At the time I saw her here, there was no evidence of renal failure. I think it is fair to say that Dr. Walter Bauer and I both saw this patient, and that I leaned toward atypical rheumatoid arthritis and he toward acute disseminated lupus.

Clinical Diagnoses

Acute disseminated lupus erythematosus?
Rheumatoid arthritis?
Pneumonia.
Terminal uremia.

Dr. Kranes's Diagnoses

Disseminated lupus erythematosus.
Terminal pneumonia.
Uremia.

Anatomical Diagnoses

Acute disseminated lupus erythematosus.
Bronchopneumonia, bilateral.
Miliary abscesses of right upper lobe of lungs.
Pericarditis, chronic, fibrous, obliterative.
Mediastinitis, chronic, fibrous.
Pleuritis, chronic, obliterative.
Splenomegaly.
Lymph-node enlargement, general.
Pulmonary congestion.
Arteriosclerosis, aortic, minimal.
Peritonitis, chronic, fibrous, localized.
Perisplenitis.
Ascites.

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Pathological Discussion

Dr. Tracy B. Mallory: The post-mortem examination, as Dr. Kranes predicted, still leaves several loopholes for doubt. The positive findings can be listed rather quickly. There was a complete fibrous obliterative pleuritis on both sides, with no fluid. The pericardium was quite markedly thickened, and Dr. Eugene Sullivan, who made the post-mortem examination, also thought that the surrounding mediastinal structures showed more fibrosis than usual. The pericardium was totally adherent to the heart, and the two together weighed about 500 gm. The right ventricle seemed to be slightly dilated and was definitely a little hypertrophied. There was no endocarditis, and no myocarditis was made out. The liver was enlarged, and showed rather marked central congestion, which merely seemed to be evidence of heart failure. The spleen was greatly enlarged, weighing 700 gm. It should have been readily palpable. The kidneys were also large, weighing a little over 400 gm. Throughout the body, all the lymph nodes were quite notably enlarged, ranging from 0.5 to 1.5 cm. in diameter. The retroperitoneal, bronchial, axillary and inguinal glands showed definite lymphadenopathy. The joints showed marked synovitis but no evidence of rheumatoid arthritis.

On microscopic examination, there is very little to add. There was a fibrous pericarditis, pleuritis, terminal pneumonia, some slight central necrosis of the liver and nothing significant about the spleen. The kidneys showed discouragingly little. An occasional glomerulus showed focal glomerulitis of a tuft, or of a portion of the tuft. They certainly did not look like the kidneys of renal failure and uremia. The convoluted tubules were a little swollen. Nowhere in the body were we able to pick up any vascular lesions that would suggest periarteritis.

We have the records of autopsies on 24 cases of disseminated lupus, and on reviewing them it is apparent that nothing is constant. Pneumonia is the commonest finding, but that is always just a terminal event and nonspecific in character. Pleuritis and pericarditis occur in the great majority of the cases. Ascites is less common, and endocarditis is uncommon. A diffuse lymphadenopathy is almost regular. Splenomegaly is quite typical. Nephritis, when one hunts very hard for minimal changes, can be found in 70 per cent of the cases. At the time of autopsy, the skin lesions could be made out in 14 cases. The existence of a real adhesive pericarditis, particularly a mediastinopericarditis, is unusual in these cases, but we have seen it and I should be strongly inclined to side with Dr. Kranes and regard this case as one of disseminated lupus despite the absence of characteristic skin lesions at any time during the disease.

Dr. Kranes: You have to make that diagnosis mostly on clinical grounds.

Dr. Mallory: Yes. We can add two more points that are consistent from the anatomic point of view: the slight changes in the kidney are of a type we see most frequently in lupus, and the changes in the lymph nodes are of the type we regularly see in lupus. Furthermore, the autopsy ruled out rheumatoid arthritis, tuberculosis and periarteritis nodosa, the only other likely possibilities in this patient.

CASE 27302

Presentation of Case

First Admission. A forty-seven-year-old laborer entered the hospital complaining of hemoptysis.

At the age of twenty-three years, the patient had had a penile chancre, and as a known syphilitic, he presented himself at the Out Patient Department seventeen months before admission for advice regarding the performance of manual labor. Fluoroscopy revealed a slight but definite enlargement of the heart in the region of the left ventricle, with a concave left upper border. The ascending aorta was prominent to the right, with increased pulsation; the hilar shadows and aortic knob were normal. On physical examination, the heart was regular at 80, with a moderately loud early diastolic murmur along the left sternal border. There was no Corrigan pulse, and the blood pressure was 205 systolic, 105 diastolic. Examination of the nervous system, as well as that of the pupils, fundi and fields, was negative. A blood Hinton reaction was positive, a lumbar puncture

normal, and the spinal-fluid Wassermann test negative. Antisyphilitic treatment was instituted, and the patient was allowed to do light work; he remained well without signs of heart failure, the blood pressure running between 205 and 140 systolic, 105 and 90 diastolic.

Six weeks before admission, the patient caught a cold in his chest, with a dry cough that finally produced small amounts of thick yellow sputum. On the day of admission, he suddenly began to cough up small amounts of bright-red blood every two or three minutes, so that at the time of observation he had raised about one and a half cupfuls. There were no accompanying symptoms and no pain.

In the hospital, there were no more hemoptyses, and on examination the patient appeared perfectly well. The fundi showed tortuous silverwire vessels, with dilatation of the veins at the points of crossing. There was a moderate pulsation of the neck vessels; the blood pressure was 180 systolic, 95 diastolic. Some confusion seemed to exist concerning the character and type of the heart murmurs. Systolic and diastolic murmurs were heard both at the apex and at the base by the various examiners in the Out Patient Department and in the hospital; however, the consensus leaned heavily toward the presence of a moderate systolic blow on the aortic area, with a moderate aortic diastolic murmur along the left sternal border. Scattered wheezes were heard throughout both lungs.

The temperature, pulse and respirations were normal.

The blood showed a red-cell count of 5,130,000 with a hemoglobin of 96 per cent, and a white-cell count of 9500. The blood Hinton reaction was positive. Examinations for tubercle bacilli in the sputum were negative.

An x-ray film of the chest showed no change, but on the fifth hospital day a line of increased density perpendicular to the diaphragm was present in the medial portion of the right lower-lung field, with a wedge-shaped area of consolidation close to it, apparently far posteriorly. No fluid was seen in the pleural cavity.

The patient's stay in the hospital was uneventful, and two weeks after admission x-ray study of the chest showed that the area of dullness had completely disappeared, so that he was discharged.

Final Admission (two years later). Although there was perhaps some slight decrease in exercise tolerance, the patient had felt well doing light work. Antisyphilitic treatment had been kept up in the Out Patient Department, but the blood Hinton reaction remained positive; the blood pressure ranged around 190 systolic, 100 diastolic. Eight months before admission, an x-ray film of the chest showed that the heart had increased slightly in size since the previous examination, the enlargement being in the left ventricle. The aorta had become slightly wider and showed a Corrigan type of pulsation.

In recent months, a to-and-fro murmur over the aortic area and down the left border of the sternum was noted frequently in the Out Patient Department, as well as an apical diastolic blow.

Three hours before admission, after a meal, the patient was seized with a sudden, severe, nonradiating substernal pain, which was constant and still present on admission. Nausea and vomiting soon followed, with rapid cyanosis and cold, clammy extremities.

On examination, the patient was well developed and well nourished, orthopneic, cyanotic, cold, clammy and sweating. The eyeballs were roaming, the pupils equal. The heart was enlarged, the apex being in the anterior axillary line. The sounds were of fair quality, with occasional extrasystoles and a diastolic murmur along the left sternal border. The pulse was barely obtainable, the rate 90, and the blood pressure 60 systolic, 0 diastolic. The lungs were clear, and the respiratory rate was 25. The abdomen was normal, and there was no peripheral edema.

An electrocardiogram showed a normal rhythm, with a rate of 90 and a PR interval of 0.15 second. There was a depressed take-off in ST_1 and ST_2, with prominent T_1 and T_2. T_3 was upright, and R_4 low, with a depressed take-off in ST_4.

The patient died eight hours after admission, without change in his physical signs.

Differential Diagnosis

Dr. Edward F. Bland: As a background for this patient's acute and rapidly fatal illness, I think there can be no reasonable doubt that he had a variable but considerable degree of hypertension and enlargement of the heart, which I am inclined to attribute mostly to the hypertension, rather than to the aortic regurgitation that was also present. The degree of aortic regurgitation seems not to have been great, certainly as judged by the blood-pressure (pulse-pressure) readings taken from time to time. Secondly, there apparently was no doubt in the minds of those who had this patient under their care that he also had cardiovascular syphilis, and I agree with that impression because he had a history of a penile lesion some twenty years before admission, a positive blood Hinton reaction, an aortic diastolic murmur, and fluoroscopically a slightly dilated ascending aorta, with increased pulsations. Although there was some confusion about the murmurs, I

think we had better rely on the interpretation of the majority, and accept the aortic diastolic murmur as diagnostic of organic aortic regurgitation, perhaps of only slight to moderate degree. The syphilis was vigorously treated.

I wonder if we might look at the x-ray films and ask the X-ray Department if they agree that syphilitic aortitis was present.

DR. JAMES R. LINGLEY: This is the first film taken, showing enlargement of the left border, which was due to left ventricular enlargement. The aorta is tortuous, and there is some bulging of the anterior portion of the ascending aorta, but the dilatation is very slight. I think that from the x-ray appearance it could be simply due to hypertension, and does not necessarily indicate syphilitic aortitis. This was the area of density described in the mid-portion of the right lower lobe — rather a hazy indefinite area extending in a wedge posteriorly into the chest wall.

DR. BLAND: Was that film taken two years before the final illness?

DR. LINGLEY: Yes.

DR. BLAND: Dr. Aubrey O. Hampton and others make the point that calcium in the ascending portion and not visible elsewhere in the aorta is strongly suggestive of syphilitic scarring. Do you see any evidence of that?

DR. LINGLEY: No. I see no evidence of calcium in the first portion of the aorta. These are later films, showing possibly a little more prominence in the region of the left ventricle; the aorta may be slightly larger, but I think it is rather doubtful.

DR. BLAND: There is certainly no saccular aneurysm.

DR. LINGLEY: No.

DR. BLAND: There is no calcified aortic valve that you can see?

DR. LINGLEY: No.

DR. BLAND: Although the x-ray films are not so helpful as I had anticipated, I think this patient probably had cardiovascular syphilis, with syphilitic aortitis; this was well treated, and the course suggests that this condition was not actually progressive. He got along very well, and there was not much evidence, either from symptoms, from signs or from interval x-ray study, of a progression in the cardiovascular syphilis. On the first admission to the hospital, two years before he died, he had had a respiratory infection prior to entry. He was raising some blood, and developed what looks to me from the x-ray description more like an infarct of the lung than pneumonia. I think this episode probably had no bearing on the later terminal acute illness.

Now we come to the last illness, the patient having had no angina, no paroxysmal dyspnea and no other cardiac symptoms in the interim. Patients with cardiovascular syphilis are notoriously apt to die suddenly either with or without preceding cardiovascular symptoms. It is difficult at first glance not to connect the two in this patient. Some die suddenly from rupture of a large saccular syphilitic aneurysm, which this patient did not have. Others die of acute coronary insufficiency, usually associated with free aortic regurgitation, indicating that the process has extended to a considerable degree about and beyond the mouths of the coronary arteries. Most of these patients, however, have some indication of acute coronary insufficiency before the sudden exitus. I do not believe this patient had free aortic regurgitation, and hence he probably did not have extensive narrowing of the coronary ostia. We can conceive that the coronary arteries were abnormally high in the aorta at their origin and perhaps being extensively involved without much indication of involvement of the aortic valve as such. I think that, however, is unnecessarily fanciful and needs no further consideration here.

How else, then, could cardiovascular syphilis suddenly cause this patient's death? It is well known that patients with syphilis may die abruptly and unexpectedly after specific intravenous treatment. This patient had been under treatment for some time without suspicious reactions, and nothing is said of very recent antisyphilitic therapy, so that I think we must assume his death was not related to therapy. In view of the foregoing remarks and with some hesitation, I shall say that I think cardiovascular syphilis had no direct bearing on his death.

What else might he have had? One must consider, at least in passing, a massive pulmonary embolus. When anyone dies in this fashion with severe pain in the chest, one has to keep that in mind. Furthermore, this man had probably had a small pulmonary infarct two years before, but I do not see that we have any reasonable evidence to attribute his death to a large pulmonary embolus. He had not been in bed. There was no infection, no obvious circulatory stasis, no swelling of the legs to indicate local vascular disease, and his veins were not engorged prior to exitus. Therefore, I am willing to discard pulmonary embolism from further consideration.

We are left with the two conditions, one of which seems to me most likely to have caused his death. First, and on the law of chances most likely, is a coronary thrombosis (myocardial infarct). The patient may certainly have had an

acute coronary thrombosis. One can work up a good case either for or against that diagnosis. Somewhat against it was the absence of preceding angina pectoris. Of course, that is not necessary. Secondly, it is a little unusual to have such severe pain as this man had beginning so abruptly; in fact, as described it was maximal at the onset. The discomfort with coronary thrombosis usually begins, as the patient often describes it, as "indigestion," which progressively increases in severity. In a few minutes to an hour or two, it becomes severe, but it is usually not maximal right from the beginning. Perhaps the description here is faulty, and I am too much disturbed by the abrupt onset. Perhaps it did start, after all, with less severe intensity and build up. However, I find other points that influence me somewhat away from this diagnosis. Ordinarily, with coronary thrombosis and myocardial infarction sufficiently extensive to end fatally in a few hours and with pain of this severity, it generally spreads somewhere else, usually to the arms, to the throat or to the back. I am bothered a little by the fact that this severe pain, if of coronary origin, remained so localized. Another point, — I am arguing against coronary thrombosis, and I think arguing myself into a hole, — the electrocardiogram is rather noncommittal. It was probably taken in the Emergency Ward, because the record is obviously from a portable machine. As stated in the record, it is surprisingly close to normal and certainly not what one would expect if one were dealing with coronary thrombosis. There is a very slight depression of ST_1 and ST_2, and no elevation of the ST interval in any lead. These are, at most, minor changes, and besides we do not know that this patient may not have been on digitalis prior to entry. Certainly, one cannot support a diagnosis with this electrocardiogram. The pulse rate was quite slow, in the absence of block, for a patient with a large myocardial infarct: it was only 90, and the degree of peripheral collapse was extreme. The drop in blood pressure, however, fits in beautifully with coronary thrombosis.

The next possibility, of course, is dissecting aneurysm. The abruptness and severity of the pain at the beginning is influencing me considerably in the latter direction. Usually, with dissecting aneurysm, the pain does not radiate to the arms, unless one of the vessels to the extremity becomes obstructed at its mouth. It often does go to the back and down into the abdomen, and even to the legs. In this patient, the pain did not radiate at all, and the blood pressure dropped rather alarmingly. Both are somewhat against dissecting aneurysm, but do not rule it out. The low pressure might be due to local disturbance of the vessels. But that is forcing the point a little. The patient continued in a critical condition and died about eleven hours after the onset. The strongest argument against dissecting aneurysm is that we believe this patient had syphilitic aortitis, and the combination is certainly most unusual. I cannot think of any other argument either for or against these two most likely possibilities, and I prefer the latter.

In closing, I suggest as the diagnoses: hypertension and hypertensive heart disease, cardiovascular syphilis (not extensive), aortic regurgitation (slight) and dissecting aneurysm of the aorta. I prefer the latter to coronary thrombosis.

Dr. William B. Breed: Would Dr. Lingley have talked very much about the aorta or aortitis if he did not know that the patient had a positive blood Hinton reaction and had been treated for syphilis?

Dr. Lingley: No. The only thing from the x-ray point of view that suggests aortitis is the fluoroscopic note of increased pulsations. From the films alone, I could not make a diagnosis of syphilitic aortitis.

Dr. Breed: Do you think the pulse rate of 90 is a fairly good argument against occlusion?

Dr. Bland: No; it is no argument either way. Do you not think that most people who have myocardial infarction of this degree, and who fail and die in the course of twelve hours, show some signs of heart failure either by a gallop rhythm or by rales at the lung bases?

Dr. Breed: I do not know that that is a good enough argument.

Dr. Wyman Richardson: I should like to ask whether most patients with dissecting aneurysm do not have an even higher blood pressure than this. It has been my experience that most of them have systolic pressures over 200.

Dr. Bland: Most of them do, but not all. Some are under 200. This patient's age does not help us either way. Of course, one can get dissection on bases other than media necrosis cystica. Dr. George M. Bartol, of the Boston City Hospital, recently demonstrated at a meeting of the New England Heart Association the case of a seventy-five-year-old man with calcific disease of the aortic valve and superimposed subacute bacterial endocarditis. This man developed between two cusps of the aortic valve a mycotic aneurysm that broke through into the middle coat of the aorta and dissected widely. There was no evidence in this case of cystic degeneration of the media.

Dr. Benjamin Castleman: But there was an infectious process.

Dr. Bland: Yes; but does it not illustrate that,

very rarely, degeneration of the media is not necessary to make possible a widespread dissection?

DR. CASTLEMAN: Years ago, it was believed that arteriosclerosis per se was the etiologic agent. The intima was arteriosclerotic, and the tear in the media occurred at the site of the arteriosclerotic plaque. We have had seven or eight such cases, but in those the dissection was limited to a short segment, not more than a centimeter or two, and did not produce any symptoms — just incidental autopsy findings.

DR. BLAND: Do you like the abruptness and severity of the onset in this case, Dr. Breed, in relation to your diagnosis of coronary disease?

DR. BREED: That does not bother me.

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CLINICAL DIAGNOSES

Coronary thrombosis.
Syphilitic and hypertensive heart disease.
Syphilitic aortitis and aortic regurgitation.

DR. BLAND'S DIAGNOSES

Hypertension and hypertensive heart disease.
Cardiovascular syphilis.
Aortic regurgitation, slight.
Dissecting aneurysm of the aorta.

ANATOMICAL DIAGNOSES

Dissecting aneurysm of aorta, with rupture into pericardium.
Media necrosis aortica cystica.
Syphilitic aortitis.
Cardiac hypertrophy.
Endocarditis, chronic, rheumatic, with stenosis, aortic.

PATHOLOGICAL DISCUSSION

DR. CASTLEMAN: The autopsy showed an enlarged heart, weighing 700 gm., and slight coronary disease without occlusion. The aortic valve showed definite interadherence of the cusps at their commissures, a finding quite characteristic of old rheumatic infection. There was no evidence of separation of the cusps to suggest syphilis. On the other hand, the aorta above the valve in the ascending portion and in the arch showed stellate scarring and linear tree barking characteristic of syphilis. This was confirmed microscopically. The ascending aorta was dilated, and measured 11.5 cm. in circumference. About 2 cm. above the aortic valve was a transverse tear in the intima. The tear was the largest we have ever seen, measuring 8 cm. across. Extending backward from the tear was characteristic dissection of the media of the aorta as far as the annulus, producing constriction of both coronary arteries, a circumstance that may cause anginal pain. As a matter of fact, the dissection must have continued into the coronary arteries because grossly there were subendocardial myocardial hemorrhages, and on microscopic examination there was adventitial hemorrhage around a few of the smaller myocardial vessels. Perhaps this was merely adventitial expansion of the blood around the coronary arteries. The dissection had not ascended above the point of intimal tear, stopping short at the point where the syphilitic aortitis became evident. The external tear had occurred into the pericardial cavity, and the patient presumably died of cardiac tamponade. As in another case we have recently discussed, there was hemorrhagic extension through the adventitia of the aorta to the adventitia of the main pulmonary artery and then into the lung. In summary, therefore, this man had rheumatic heart disease with aortic stenosis and probably some degree of regurgitation, syphilitic aortitis and a dissecting aneurysm of the aorta, with rupture. Sections of the aorta showed in addition to the syphilis a diffuse mucoid degeneration of the media, and in a few areas there were suggestive cyst formations. The cause of the dissecting aneurysm was most certainly the medial necrosis, and the syphilis was probably only an incidental finding. It is quite possible that the syphilitic scarring prevented the distal extension of the dissection.

DR. BLAND: Was the mitral valve all right?

DR. CASTLEMAN: Yes.

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ANNUAL MEETING OF THE AMERICAN MEDICAL ASSOCIATION

The annual meeting of the American Medical Association taxed the capacity of the hotels at Cleveland and even filled to overflowing a large lake steamer moored at the docks. It should be noted that the House of Delegates finds it an increasingly difficult problem to select cities capable of handling this tremendous gathering of physicians — hotel accommodations must be adequate, and at the same time, sufficient meeting places must be available for the House of Delegates, the various sections and the scientific and commercial exhibits. In spite of the crowding, the meeting was an outstanding success. The scientific exhibits were remarkable in their completeness and arrangement, and the papers were up to their usual standard of excellence. The Cleveland Public Auditorium proved adequate for the meetings of the sections and for the exhibits.

The general atmosphere of the House of Delegates was saturated with preparedness, approached from military, social and economic angles. President Nathan B. Van Etten, in his address to the House, again urged a department of health at Washington, headed by an officer of Cabinet rank, a suggestion that, up to the moment, has received but little sympathy in Washington. President-elect Frank H. Lahey stressed the necessity of subordinating all trivialities and conflicts of opinion to the pressing need of national unity and preparedness.

The Committee on Preparedness, appointed at the meeting in New York City last year, presented through its chairman, Dr. Irvin Abell, a lengthy report detailing its activities. This report is presented in full in the June 21 issue of the *Journal of the American Medical Association* and should be read carefully by all physicians. It stressed particularly the deferment of medical students and interns by local draft boards, so that a constant supply of well-qualified physicians should continue. On recommendation of this committee, the House later adopted a statement to be sent to the Secretary of War and the Surgeon General of the Army urging the War Department to provide some form of military status for all students in approved medical schools and for all premedical students accepted for enrollment in approved medical schools, to make clear that in preparing themselves to become physicians such men are not trying to evade their responsibilities or the draft.

The Committee also recommended, and the House adopted, a resolution urging the creation by the Government of a central authority for the procurement of physicians for assignment to the United States Army, Navy and Public Health Service and to the civilian and industrial needs of the country. This agency would contain in its membership representatives from the civilian medical profession, and would have authority to determine who should be called to service and

who should stay at home. Although the Committee on Preparedness has the machinery to prepare lists of available physicians and is able to evaluate their capabilities and, in some measure, their necessity at home, it has no power to enforce its judgment; neither has the Government at the present time. This authority would have some power, and should function so that whole communities would not be left without adequate medical care.

A certain amount of unrest among many physicians in regard to the various specialty boards was brought into the open by two resolutions presented to the House. The first proposed a committee from the House of Delegates to confer with the various boards, and the second that efforts be directed toward creating a board of general practitioners. Neither resolution prevailed. It was the consensus of the delegates that the boards were independent of the American Medical Association, being more or less controlled by the Advisory Board for Medical Specialties.

The House of Delegates, in executive session, listened to a résumé of the trial of the American Medical Association and certain of its constituent societies in Washington and voted, without a dissenting vote, to authorize the Trustees to appeal the verdict to the Court of Appeals of the District of Columbia and, if necessary, to the Supreme Court of the United States, so that it shall once and for all be determined whether medicine is a trade or a profession.

A significant feature was the designating of the 1942 meeting at Atlantic City as the Pan American Session, to which the South American medical societies will be asked to send special representatives.

The free and open discussion, the careful thought given by the reference committees to each resolution and the high percentage of attendance at the meeting speak well for the quality of men selected by the constituent state societies as delegates to the House of Delegates. This is as it should be, since the House of Delegates is the only body that has the power to determine the policies of the American Medical Association.

THE EPIDEMIOLOGY OF CANCER

ALTHOUGH, in its narrower interpretation, the term epidemiology can hardly be applied to a noninfectious disease such as cancer, nonetheless the methods applicable to the study and control of epidemic diseases are so serviceable in advancing the knowledge of cancer and its control that the utilization of the term in this broader sense is perhaps justifiable. The epidemiologist deals with disease processes in mass and is concerned not so much with the problems of the care of the patient as with learning all he can regarding the etiology, predisposing factors, nature and prevalence of the sickness. With these in mind, means of prevention and means of control may, at least to a limited degree, be worked out.

The first problem to be settled with regard to cancer is that of its incidence. Work has been done in this field for many years, and modern mortality statistics are fairly accurate. But even yet there is very little useful information regarding the morbidity of cancer. As more and more cases are cured, and as the different types of the disease have means of therapy developed for them, it becomes of increasing importance to know as many as possible of the details of incidence. In several localities, morbidity studies are now being carried out, and it is hoped that much may be learned from them.

The increasing accuracy of mortality statistics and the mass of data accumulated in hospitals have already demonstrated many things about cancer. Although it was once considered a disease of old age, it is recognized that certain types occur early in life, and some, such as tumors originating from nerve cells, are almost entirely restricted to that period. In general, it is true that the prevalence of cancer rapidly increases with the advance in years. Sometimes a given type of cancer, such as osteogenic sarcoma arising in bone, may have two different peaks of incidence in relation to age — one in adolescence and the other in old age. The occurrence of this tumor in old age is clearly correlated with a predisposing condition — Paget's disease in the bone. From these few examples, it may be seen that cancer is not one disease, but

rather many diseases different in their courses, in their modes of treatment and, presumably, in their causation.

Some years ago it was noted that workers in certain of the aniline-dye industries developed cancer of the bladder with unusual frequency. By careful observation of these cases, and comparison of the incidence of bladder cancer with that of the general population and with that of workers in other portions of the industry, it was finally found that one particular substance — naphthylamine — was the responsible factor, and that by preventing exposure to it this particular hazard could be overcome.

Still another example of special causation of a particular type of cancer is afforded by a group of miners at Schneeberg, in Austria, who worked in a mine where the ore was radioactive; because of the inhalation of radioactive particles over a period of years, many of these men developed cancer of the lung. Furthermore, the early workers in x-ray and radium provided excellent, though unfortunate, examples of the potential danger of these useful diagnostic and therapeutic tools, since the toll of skin cancer among this group of workers was exceptionally high.

In the past year or two, it has begun to be realized that an industrial hazard only recently thought to be fairly clearly understood might also produce neoplastic disease. The production of severe anemia by benzol poisoning has been known and recognized for some time, but the fact that with slight exposures a hyperplastic response of the marrow, leading to the development of leukemia, might occur has only recently been appreciated.

Race is a significant factor, but one difficult to evaluate, since many aspects of environment, habit and diet may be associated with it. The rarity of cancer of the cervix in Jewish women and the frequency of carcinoma of the stomach among Scandinavian races are examples of this type of race selectivity. Still another complicating factor in an understanding of the incidence of malignant disease is the existence of individual susceptibility. It has been clearly demonstrated that the occurrence and cure of one cancer confer no immunity, but rather that there is a definite susceptibility of a person who has one cancer to the development of a second, or even of a third. These matters of racial and individual susceptibility are most troublesome when it comes to evaluating statistical material.

Certain lesions, some inherited and some acquired, are clearly recognized as precancerous. Thus, the occurrence of benign polypoid growths of the large intestine is an inherited abnormality, and such growths are a very frequent source of cancer of the large intestine. In the same way, certain acquired abnormalities subsequently lead to the development of cancer. Thus, there is a greater proportion of skin cancer in the total of all cancer in the South and Southwest than there is in New England, apparently directly ascribable to a greater exposure to sunlight, heat and dust. The woman who develops cystic disease of the breast develops a precancerous lesion. This type of disease process, quite prevalent, has been regarded by some as harmless, by others as a definite precursor to cancer. Only recently has a clear-cut answer to this problem been reached. On the basis of a large number of women followed for an average of over nine years, it was found that those women who developed chronic mastitis before the menopause had eleven and a half times as much cancer of the breast as the normal population did; after the menopause the difference was less striking — two and a half times as much. Furthermore, it is practically axiomatic that a cancer never develops in a normal mouth. Instead, one finds as predisposing factors such changes as leukoplakia, syphilis of the tongue and chronic irritation from bad teeth.

Progress will continue, if, as one learns more regarding the varied aspects of cancer, the disease is studied by epidemiologic methods, rather than by considering each case as an isolated problem. Means of prevention of certain types of cancer are already available. Methods of therapy are steadily improving, and their early application will lead, even without further advances in knowledge, to a marked lowering of the cancer death rate.

MEDICAL EPONYM

Jacksonian Epilepsy

This condition bears the name of John Hughlings Jackson (1834–1911). The following quotation was written by an anonymous contributor to the column, "Reports of Medical and Surgical Practice in the Hospitals of Great Britain," and appeared in the *British Medical Journal* (**1**: 773, 1875), under the title, "London Hospital: Clinical memoranda of a series of interesting cases of nerve-disorder now in hospital (under the care of Dr. Hughlings Jackson). . . ."

> . . . . In the convulsions spoken of (commonly called epileptiform convulsions), a good deal occurs before the patient loses consciousness. One patient gave a very vivid account of what Dr. Hughlings Jackson calls the "march of the spasm." This patient's fit begins in his left index-finger and thumb; it then passes up the arm, and affects the face, and next passes down the leg. It is the rule that fits which begin in the hand should begin in the index-finger and thumb; when they begin in the foot, they usually begin in the great toe.
>
> Speaking of these cases, and with reference to their difference from such cases as are commonly called epilepsy par excellence, Dr. Hughlings Jackson said that he thought the abrupt division into cases with and cases without loss of consciousness was not even justifiable on grounds of convenience. . . . The distinction was, he insisted, into cases where consciousness was lost first of all, very early or late in the paroxysm.
>
> R. W. B.

MASSACHUSETTS MEDICAL SOCIETY

SECTION OF OBSTETRICS AND GYNECOLOGY*

Death Associated with Peritonitis Following Cesarean Section

A thirty-seven-year-old para VII had consulted no physician until labor started. Shortly after the onset of labor, the patient began to bleed from the vagina and was sent into the hospital. The membranes ruptured shortly after arrival in the hospital, at 6:35 p.m., and labor ceased. The hospital notes state that there was a good deal of bleeding during the night, and that at 3:30 the following morning a consultation was held. No diagnosis was made, and a cesarean section was performed. The pulse at that time ranged from 140 to 150. When the abdomen was opened, the baby and the placenta were found free in the abdominal cavity. The uterus had apparently contracted, and there was no evidence in the note that the uterus had ruptured. The diagnosis of the operator was abdominal pregnancy, and the abdomen was closed. The patient was not transfused. She died three days after operation, apparently of peritonitis.

Comment. The record on this case is very incomplete. The past history mentioned only that she had had no evidence of cardiac or renal disease. The blood pressure was not recorded, and the only reference to the pulse was that before operation it had risen to 150. Although the death certificate attributes this death to abdominal pregnancy, such a diagnosis is extremely unlikely. Abdominal pregnancies are not characterized by excessive vaginal hemorrhage. The large amount of external bleeding in this case suggests that the placenta had been attached to the uterus, and the fact that the baby and placenta were reported to have been found free in the abdominal cavity, the placenta being without any attachment, bears out this diagnosis.

The observation that shortly after 6:35 p.m. labor ceased but hemorrhage continued is interesting. It is perfectly possible that, when labor ceased, rupture of the uterus occurred. Uteri do spontaneously rupture at term. An operation, followed by transfusion, when this patient arrived, after an intelligent diagnosis had been made, would probably have saved her life. To avoid such disasters, patients must be followed during pregnancy. Those financially unable to have private physicians must be taken care of by some sort of clinic. Without prenatal care, there can be no personal relation between patient and obstetrician, which is a vital part of successful obstetrics. It is difficult to understand how this patient, who was reported to have been bleeding very freely by vagina at 6:35 p.m. and who continued to bleed for nine hours, passing clots, had no vaginal examinations during this time for the purpose of establishing an intelligent diagnosis. Had an abdominal pregnancy existed, which is unlikely, and had it been diagnosed at 6:35 p.m., an operation at this time would probably have saved the patient's life.

This death can be attributed to either ignorance or indifference on the part of the medical attendant. A higher sense of responsibility throughout the profession at large can be acquired only by improved education in medical schools, by additional training in a hospital after graduation and by a greater sense of responsibility on the part of the hospital authorities so that they will demand of those physicians practicing in their institutions

*A series of selected case histories by members of the section will be published weekly. Comments and questions by subscribers are solicited and will be discussed by members of the section. Letters should be addressed to Dr. Raymond S. Titus, *Secretary*, 330 Dartmouth Street, Boston.

a higher standard of obstetric routine. In this column, it has frequently been emphasized that hospital trustees are ultimately responsible for both good and bad medicine, and good and bad obstetrics, as practiced in their institutions. They have the power to regulate obstetrics in their hospitals, and until supervised obstetrics is practiced in all institutions, such fatalities as this will be repeated.

INFANTILE PARALYSIS*

Almost every summer, when infantile paralysis breaks out in some section of the country, there is an extraordinary degree of apprehension. This is not so much because of the large number of cases, as compared with epidemics of other diseases, but because of the fear of paralysis. Then, too, the unexpectedness with which the disease strikes and the uncertainty as to where it will appear add to the alarm. This feeling of helplessness is responsible for a pressing demand for more knowledge about the disease. As a result, infantile paralysis has been studied much more closely than many of the far commoner diseases to which we seem to have become more accustomed.

In many diseases, the cause is quite apparent from some striking circumstance; for example, the connection with the sick parrot in psittacosis and with the dog-bite in rabies and the exposure to a case in smallpox or measles or to the carrier in typhoid fever. But in infantile paralysis, no one circumstance immediately tells us which way to turn in attacking the problem. Any theory becomes a subject of study. Through the years, a great variety of seemingly contradictory findings have accumulated. Moreover, the clinical disease is sparsely distributed, and the available tools to study it in the laboratory are so restricted that studies in the field or experiments in the laboratory have often been limited to such small numbers that it has been difficult quickly to recognize the important or to rule out the unsound theories. Since it is difficult to discriminate between significant and insignificant observations, it is not easy to harmonize the various findings into a convincing concept. The result has been that we find all sorts of conflicting hypotheses, often based on small bits of evidence, and with seeming preference for those that have a more hopeful sound or match in oddness the vagaries of the paralytic disease. If, instead of forming a new theory every time a new bit of evidence is collected, the many pieces of evidence are fitted together like the jigsaw puzzle, we can begin to see a picture that could hardly be imagined from even the closest examination of the pieces separately. From the maze of collected studies, a few facts stand out clearly, facts that could not be discovered from the study of individual cases or epidemics, or from any single experiment.

The dissemination of the virus far exceeds the occurrence of the frank disease. Evidence that the virus is generally and rather uniformly widespread is seen in the geographic prevalence of the disease — we find it all over the globe; in the difference in the ages at which it occurs in city and country populations; and the finding that many persons become immunized who have never had any symptoms of the disease. In striking contrast, the occurrence of the paralytic disease is restricted to a small fraction of those exposed to the virus.

Many who come in contact with the virus show no symptoms. More of the milder forms of the disease are now being recognized, so that a diagnosis of infantile paralysis no longer means, as it did a few years ago, inevitable paralysis. And we are beginning to ask ourselves when we see these primary symptoms: Will this be infantile paralysis, or is it merely a harmless infection with its virus, trivial in itself, but taking its full part in passing the virus along and keeping it in circulation?

Now that we understand that this virus, which is so widespread, — perhaps as widespread as measles, — is harmless to the vast majority of people, we might do well not to concentrate all our efforts toward the probably impossible task of keeping it from spreading, but rather to study the circumstances that, in addition to exposure to the virus, make for the occurrence of paralysis in the exceptional few of those exposed. There are reasons for believing that a difference in those exposed, rather than any difference in exposure, determines whether or not paralysis occurs. Paralysis picks not just any who are exposed, but a certain few. Some of these selectivities may give a clue of the cause of this susceptibility to paralysis, and this appears to be one of the most important things to learn about the disease, because paralysis is the one serious consequence of infection with this virus.

What are the selections that govern paralysis? The frequency of paralysis in those exposed varies in different climates, and also with season in the same locality. There are many persons who are exposed without having the recognizable disease in regions nearer the equator, and this is also true in off-seasons in temperate zones, where infantile paralysis is commoner. This influence of climate and season on the prevalence of the disease suggests that some physiologic process, which fluctuates with climate and season, conditions the response of persons to exposure to the virus.

A reason for believing that some factor in the patient determines the development of the disease on exposure to the virus is its tendency to occur in multiple numbers in some families, not only in brothers and sisters, but in members of successive generations, and in both lineal and collateral lines. This familial frequency is out of proportion to the low incidence of the disease in the general population, and suggests that susceptibility is in some way associated with an inherited quality.

That the disease tends to occur in persons of a certain constitutional type has long been suspected by those who see many cases. The observation, although by no means universal, has nonetheless been made often enough to suggest that children, whose glandular functions regulating growth and development are in some way modified, are more susceptible to paralysis than others.

Again, the virus of infantile paralysis evidently enters the system through the mucous membrane of the upper respiratory tract, and this mucous membrane, when sound, is probably the barrier that prevents the virus from reaching the nervous system. A number of cases of paralysis have followed tonsil and adenoid operations, and this bears out the idea that injury to mucous membranes may let the virus through to the nervous system.

Moreover, there is an impression that infantile paralysis occurs oftener than would be expected on the laws of chance in persons at certain times, when there are known

*A "Green Lights to Health" broadcast given through Station WAAB by Dr. W. Lloyd Aycock on Wednesday, April 30, and sponsored by the Public Education Committee of the Massachusetts Medical Society and the Massachusetts Department of Public Health.

changes in the mucous membranes due to alterations in hormone secretion. These naturally occurring changes have been reproduced experimentally in monkeys. When these animals are given instillations of the virus in their nostrils, their resistance is seen to be different from that of normal animals. In addition to these experiments, it has been found that children who have had infantile paralysis have different glandular secretions as compared with those who have not had the disease.

Thus, from all these studies, we may say that the exceptional occurrence of paralytic infantile paralysis among the many who are exposed to the virus is determined by individual susceptibility, which is physiologic in nature and bound up with differences in certain of the endocrine glands. Infantile paralysis, therefore, does not present the simple problem of a disease to which we can apply universal preventive measures, such as smallpox, in which we can vaccinate everybody, or rabies, in which we can readily select the few who need vaccination — those bitten by a rabid animal. From the point of view of paralysis, infantile paralysis is a disease of limited occurrence, and close attention must be paid to its behavior when the practicability of preventive measures is considered.

These talks are called "Green Lights to Health." The "green lights" in infantile paralysis now seems to be in the direction of studying the question of susceptibility, if we are to be able to pick out the few who are susceptible, to try to understand what makes them susceptible, and to learn how to correct their susceptibility in the hope of turning what is for them a distressing disease into what is for most people a harmless immunizing infection. For it may not be feasible to adopt too sweeping or drastic measures for the benefit of a few of the many who are exposed to the virus.

Now that knowledge has extended to the milder infections with this virus, we can take some comfort, if an epidemic appears, when we remember that over half the cases diagnosed do not develop paralysis, that probably half the patients who do develop paralysis recover entirely, and that many of these are so slightly affected that they are not at all handicapped in following any of the ordinary pursuits of life.

DEATHS

CHRYSTAL — Michael H. Chrystal, M.D., of Leominster, died July 13. He was in his seventy-fourth year.

Born in North Adams, Dr. Chrystal received his degree from Baltimore Medical College in 1901. He was a member of the surgical staff of the Leominster Hospital, and was a fellow of the Massachusetts Medical Society and the American Medical Association.

His widow and two sisters survive him.

SMITH — Alfred C. Smith, M.D., of Brockton, died July 15. He was in his seventy-third year.

Born in Capetown, South Africa, Dr. Smith received his degree from Tufts College Medical School in 1898. He was a former president and treasurer of the Plymouth District Medical Society, and a member of the Massachusetts Medical Society, being a member of the Committee of Ethics and Discipline for many years, and of the American Medical Association.

His son survives him.

NEW HAMPSHIRE MEDICAL SOCIETY

DEATH

CHEEVER — Nathaniel F. Cheever, M.D., of Greenfield, died June 15.

He was born in Walden, Vermont, in 1856, graduated from the University of Vermont Medical College in 1883, and had practiced in Greenfield and the surrounding towns since 1884. He was a member of the American Medical Association and of the New Hampshire Medical Society, was prominent in fraternal organizations and held various town offices. He represented Greenfield in the Legislature in 1909–1911.

KITTREDGE — Frank E. Kittredge, M.D., of Nashua, died July 8. He was in his eightieth year.

Born in Concord, Dr. Kittredge received his degree from the University of Pennsylvania School of Medicine in 1885. He had practiced in Nashua since 1889 and was one of the founders of the Nashua Memorial Hospital. He was a member of the New Hampshire Medical Society, the American Medical Association, the American College of Surgeons, the American Academy of Ophthalmology and Laryngology and the American Laryngological, Rhinological and Otological Society.

MISCELLANY

RÉSUMÉ OF COMMUNICABLE DISEASES IN MASSACHUSETTS FOR MAY, 1941

Diseases	May 1941	May 1940	Five-year Average*
Anterior poliomyelitis	0	1	3
Chicken pox	1277	1207	1167
Diphtheria	9	11	17
Dog bite	1284	1373	1271
Dysentery, bacillary	2	15	9
German measles	640	109	315
Gonorrhea	319	313	393
Lobar pneumonia	209	386	414
Measles	4117	4058	3876
Meningococcus meningitis	8	3	14
Mumps	1479	943	1047
Paratyphoid B fever	22	22	25
Scarlet fever	827	670	965
Syphilis	437	518	493
Tuberculosis, pulmonary	333	350	274
Tuberculosis, other forms	34	31	26
Typhoid fever	9	16	6
Undulant fever	12	8	4
Whooping cough	1158	757	684

*Based on figures for preceding five years.

Geographical Distribution of Certain Diseases

Diphtheria was reported from: Acton, 1; Boston, 2; Brookline, 1; Cambridge, 1; Fall River, 2; Revere, 1; Watertown, 1; total, 9.

Dysentery, bacillary, was reported from: Waltham, 1; Worcester, 1; total, 2.

Malaria was reported from: Winthrop, 1; total, 1.

Meningococcus meningitis was reported from: Brockton, 2; Chatham, 1; Fall River, 1; Hardwick, 1; New Bedford, 1; Quincy, 1; Taunton, 1; total, 8.

Paratyphoid A fever was reported from: Charlton, 1; total, 1.

Paratyphoid B fever was reported from: Cambridge, 1; Foxboro, 1; Lexington, 9; Malden, 8; Tewksbury, 1; Waltham, 1; Worcester, 1; total, 22.

Pellagra was reported from: Boston, 1; total, 1.

Septic sore throat was reported from: Andover, 2; Beverly, 1; Boston, 8; Fall River, 1; Gill, 1; Merrimac, 2; Milton, 1; Oxford, 1; Scituate, 1; Williamstown, 1; total, 19.

Tetanus was reported from: Dudley, 1; Middleboro, 1; New Bedford, 1; total, 3.

Trachoma was reported from: Boston, 1; total, 1.

Typhoid fever was reported from: Arlington, 1; Boston, 4; Carver, 1; Leicester, 1; Medford, 1; Newton, 1; total, 9.

Undulant fever was reported from: Bridgewater, 1; Gloucester, 1; Leominster, 5; Milford, 1; Newton, 1; Pittsfield, 1; Shelburne, 1; Worcester, 1; total, 12.

German measles, measles, mumps, pulmonary tuberculosis, undulant fever and whooping cough show an increased prevalence, all being higher than the average for the five previous years.

Diphtheria, bacillary dysentery, lobar pneumonia and typhoid fever were reported less frequently than usual, all except the last being below the five-year average.

No cases of anterior poliomyelitis were reported during the month.

Meningococcus meningitis has been gradually increasing, but has not as yet reached the five-year average.

The incidences of chicken pox, dog bite, paratyphoid fever and scarlet fever were at about normal levels.

NOTES

The following promotions on the faculty and teaching staff of the Harvard Medical School, effective next September, were recently announced: Donald L. Augustine, associate professor of comparative pathology and tropical medicine; William L. Aycock, associate professor of preventive medicine and hygiene; Harry C. Solomon, clinical professor of psychiatry; Charles G. Mixter, clinical professor of surgery; Robert S. Morison, assistant professor of anatomy; Thomas D. Jones, assistant professor of medicine; Aubrey O. Hampton, assistant professor of roentgenology; Arthur T. Hertig, assistant professor of pathology; Jacob Fine, assistant professor of surgery; Harold G. Tobey, lecturer on laryngology; Henry R. Viets, lecturer on neurology; Charles T. Porter, lecturer on otology; George G. Smith, lecturer on genitourinary surgery; Henry S. Bennett and Edward W. Dempsey, associates in anatomy; John L. Oncley, associate in physical chemistry; Mark D. Altschule, Howard F. Root and Eugene A. Stead, Jr., associates in medicine; Charles D. May, associate in pediatrics; Eric Lindemann, associate in psychiatry; John E. Dunphy, Franc D. Ingraham, Champ Lyons, Leland S. McKittrick and Augustus Thorndike, Jr., associates in surgery; and John H. Harrison and Fiorindo A. Simeone, associates in genitourinary surgery.

President Leonard Carmichael of Tufts College has recently announced that a fund, representing a fiftieth birthday present from over two hundred friends to Mr. Joseph F. Ford, a Boston manufacturer, has been turned over to Tufts College Medical School. It will be known as the Joseph F. Ford Student-Aid Fund, and the income will be used to help deserving students to defray the costs of medical education.

CORRESPONDENCE

"PRACTICAL ASPECTS OF SURGICAL SHOCK"

To the Editor: Dr. J. Englebert Dunphy's review of the shock problem in the May 22 issue of the *Journal* is neat and timely, but it would be unfortunate if his article were to spread an impression that observation of the pulse and of the arterial blood pressures fails to detect shock early.

Pulse rates were of no value, he and his collaborators have concluded in recently published experiments with barbiturized animals. Their published protocols show fast control pulse rates, often 100 to 130. Many physiologists use only well-trained animals, to avoid the unnatural experimental conditions of narcosis, and they have shown the normal pulse rate of large dogs to be 60 to 80. Barbiturate narcosis has been shown to raise the pulse rate through sympathetic stimulation. But in any case, the protocols of Dr. Dunphy and his collaborators show rather immediate further elevation of pulse rates of burned or traumatized barbiturized dogs to maximums of 150 to 200 beats a minute. Even in barbiturized dogs, then, the pulse rate seems to react definitively in the early stage of shock. Their conclusion has no basis in fact: let them first show dogs approaching shock with pulse rates below 80.

More unfortunate is their error of calling the pressure recorded by an intra-arterial Ludwig mercury manometer "the blood pressure" in their text and "systolic pressure" in their tables. It is only the integrated mean arterial pressure, that is to say, the mathematical average of the changing pressures in a given artery during the whole heart cycle. Of course they, like everyone else, find that this mean pressure is well maintained throughout early shock, in the compensated stage of "diminished effective blood volume." The same sort of maintenance of mean pressure takes place in us every day when we rise from the lying to the standing position: at the same time the venous return and therefore the cardiac output diminish, the pulse rate rises very slightly, and the pulse pressure (systolic arterial minus diastolic arterial pressure) narrows definitely. Every physician who understands what he is studying when taking a Schneider index knows that he must observe the diastolic arterial pressure and that he should consider the systolic only in relation to it: he speaks of the blood pressures, not of the blood pressure.

Experimentally, investigators of the shock problem must discard the hoary Ludwig mercury column and must learn from physiology texts the meaning of diastolic arterial pressure. The use and necessity of a Huerthle spring-type or better a Hamilton membrane-type manometer deserve stress. It is easy to demonstrate with one of these that as soon as a small fraction — 5 to 10 per cent — of the blood volume has been withdrawn, the pulse pressure narrows, usually with a distinct rise in diastolic and maintenance of mean arterial pressure. When small fractions are repeatedly withdrawn, the pulse pressure becomes progressively narrower and finally the mean pressure fails. This simple fact holds both in trained unanesthetized and anesthetized animals. It can be observed in any blood donor. I believe the experimental surgeons' persistent error in using Ludwig manometers should be called to account. Their continued inadequate concept and their thinking in terms of "the blood pressure" should no longer merit attention.

Experimental surgeons have slowly and laboriously gained a fruitful concept of "effective blood volume." This is qualitative at best. Now they urge universal adoption of hematocrit readings and red-cell counts and of falling-drop and plasma-protein determinations. These factors, in different stages and in different types of shock, change in varied directions. They, also, are qualitative when it comes to being interpreted. The surgeons still have to emphasize, perfectly correctly, that "correct appraisal of the condition [of the patient in shock] and the institution of proper replacement therapy are dependent on a careful study of the patient as a whole." Since the laboratory determinations which they recommend are not always reliably done even under the hothouse conditions of the experimental laboratory, and since they must be evaluated in a qualitative manner even by the expert, I urge a wider appreciation of the simple, mean-

ingful and quantitative determinations of the pulse rate, and of the pulse pressure and the diastolic arterial pressure — the systolic pressure and the mean pressure are best forgotten. These measurements objectively check the all-important clinical observation of the patient, especially his tongue, and the warmth and moistness and color and pulse quality of his extremities. Unlike hematocrit readings and plasma-protein determinations, they are available to every practitioner and in every circumstance, civil and military.

Physiologists have striven to avoid the qualitative and shifting sands of "effective blood volume," albeit they neglect "hematogenic, vasogenic and neurogenic" types of shock. They do not speak of "effective blood volume," but they quantitatively measure cardiac output. They show that during compensated shock the mean arterial pressure is well maintained, but the venous pressure and venous return (or cardiac output) are diminished. They teach that this measurable diminution, not the late drop in mean pressure of uncompensated shock, is the essential pathologic change. Pulse rate times pulse pressure gives an index which is a good serviceable first approximation of the cardiac output. No method of measuring cardiac output is ideal, not even the gas-absorption methods or Hamilton's dye method. This last is to be recommended, nevertheless, in all investigations that include blood-volume studies, for it can be combined with them easily without loss of accuracy to either measure. For clinical use, a diminishing series of products obtained by multiplying pulse rates by pulse pressures is an early, accurate measure of diminishing effective blood volume and impending shock. It can be interpreted in only one way, unlike hematocrit readings and plasma-protein determinations; it applies to shock of every stage and of every type; and it requires only an attending physician with a watch and a sphygmomanometer.

Future study of shock ought to elucidate in what parts and to what degree the circulation is inadequate. The body extremities can be studied simply with oncometers, but conditions are easily vitiated by changes of body temperature. Visceral circulation studies are very painstaking, and require trained unanesthetized animals and Rein's *Thermostromuhr*. These methods will teach us just how much various parts of the body are made to sacrifice their needed circulation to the end that the delicate brain cells and their jealous guardian, the carotid sinus, receive a proper supply of oxygen and glucose; perhaps the kidney will be found to assert itself through its chemoregulators. As yet, we are ignorant of how much and for how long the various parenchymatous and vascular tissues can undergo oxygen deprivation and other metabolite insults. Real understanding of shock cannot come in advance of such studies.

Meanwhile the understanding observation of pulse rates and of blood pressures, both pulse and diastolic, deserves not less but rather more stress. If these objective measures have not been helping all of us, the fault is not in them but in ourselves.

ROBERT J. KINNEY, M.D.

512 East State Street
Ithaca, New York

* * *

The above letter was submitted to Dr. Dunphy, and the following reply was received:

To the Editor: Theoretically it is true, as Dr. Kinney points out, that a diminishing series of products obtained by multiplying pulse rate by pulse pressure is a measure of diminishing effective blood volume and impending shock. Perhaps it deserves more rather than less attention.

However, the title of my review was "Practical Aspects of Surgical Shock," and in actual practice, determinations of the pulse rate and pulse pressure are quite unreliable as a guide to fluid requirements in individual patients. To take a practical example: during the course of some recent studies, 1220 cc. of blood was removed in thirteen minutes from a volunteer subject. This constituted an actual reduction of the blood volume of over a quarter of the estimated total. Before removal of the blood, the pulse rate was 60, and the blood pressure 114 systolic, 75 diastolic. Four minutes after removal of the blood the readings were pulse 84 and blood pressure 110 systolic, 80 diastolic. By no stretch of the imagination could these figures, or even the difference between these figures, be construed as evidence of a serious reduction of the blood volume. To a surgeon, however, the loss of over a liter of blood may be of considerable importance, and he must attempt to anticipate and correct such losses before significant alterations of the pulse and blood pressure occur.

This experiment is not a theoretical or hypothetical problem. The same thing occurs daily in surgical practice. To cite another example: a patient recently entered this hospital, seventeen hours after a free perforation of a duodenal ulcer. At the time of admission he was in evident peripheral vascular collapse. The pulse rate was 140, and the blood pressure could not be obtained. Following the administration of morphine and 1000 cc. physiologic saline solution in the Emergency Ward, the pulse rate was 110, and the blood pressure 105 systolic, 70 diastolic. Now a pulse rate of 110 and a blood pressure of 105 systolic, 70 diastolic, are not in themselves indicative of shock. Such levels are commonly observed under Avertin or spinal anesthesia without having any grave prognostic significance. Therefore, it is important that the hematocrit reading in this particular patient was 75 per cent and the serum protein 4.5 gm. per 100 cc., indicating an enormous reduction of the blood volume. Nearly 2000 cc. of blood and plasma were required to restore this patient's hematocrit reading and serum-protein level to normal, at the end of which time he had improved to such a degree that he withstood operation without difficulty. Over 2000 cc. of fluid with a high protein content (3.8 gm. per 100 cc.) was found in the peritoneal cavity. Is a pulse rate of 110 and a blood pressure of 105 systolic, 70 diastolic, indicative of such an enormous loss of fluid?

In closing, it is important to emphasize that determinations of the hematocrit and the plasma protein are quite simple procedures, readily done by any physician or technician capable of doing a red-cell count. The only expensive equipment needed is a good centrifuge. Finally, it should be stressed, as I emphasized in my review, that no laboratory procedures can supplant an accurate and careful clinical appraisal of the patient in shock. Examination of the pulse and blood pressure are procedures that will never be discarded and that often provide information of great value, but it is no longer correct to consider them infallible guides to the state of the circulation.

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REPORT OF MEETING

NEW ENGLAND PEDIATRIC SOCIETY

At a meeting of the New England Pediatric Society, held on April 30 at Longwood Towers, Brookline, Dr. Samuel Levine, professor of pediatrics at Cornell Medical College, spoke on "Some of the Physiologic Peculiarities of the Premature Infant."

Dr. Levine began his talk by stating that the transition from intrauterine to extrauterine existence demands adaptations, and that premature infants make these adaptations less successfully, owing to their inadequate physiologic development. Charts were then shown of the causes of infant deaths: prematurity led, with 47 per cent, and birth injury and congenital malformations followed, with 14 and 10 per cent, respectively. The great preponderance of prematurity over any other single cause of infant death left little more to be said about the importance of the topic of discussion. It was further pointed out that even more significance should be attached to infant deaths as a whole, because of the present decline in birth rate.

The first physiologic system discussed by Dr. Levine was the respiratory apparatus. In this respect, it was shown that the premature infant was heavily handicapped, since five other systems may contribute to respiratory distress: the nervous system, by having a poorly developed gag and cough reflex; the vascular system, by decreased development of capillary circulation and thus more danger of atelectasis; the skeletal system, by weakness of the thoracic cage; the blood, because lowered fetal hemoglobin gives poor oxygen intake; and finally, the connective tissues, owing to lack of development of elastic tissues in the pulmonary vessels. The chemical stimulation of respiration in the premature infant is less sensitive than in the fully developed infant, the speaker reported; he showed a series of charts of his experiments in varied oxygen and carbon dioxide mixtures, which supported his statement.

Another very serious handicap to the premature infant, as compared with the full-term infant and the infant a few days after birth, was shown to be the delayed development of the capillaries of the medulla and lungs. Many slides were shown in which the capillaries, clearly demonstrated by the injection of dyes, were markedly inadequate in the medullae and lungs of premature infants. A well-graded series of these slides illustrated the speaker's next contention — that the capillary development of the lungs and medulla is proportional to the birth weight of the baby. The elastic tissue in the blood vessels was also found to be proportional to the birth weight of the infant.

The subject of bodily temperature was then taken up, and here again the premature infant was said to be badly handicapped because of a combination of several factors. Owing to incomplete muscular and systemic development, there is, first of all, low total-heat production. This is enhanced by the slow metabolism and consequent general inactivity of the infant. Inadequate use of calorigenic hormones (thyroxin and adrenalin) was also reported in premature infants, as well as poor sweating mechanisms on exposure to heat. One of the chief difficulties in heat regulation met by the underdeveloped infant was said to be due to the lack of proportion of surface area to body weight, the surface area being in considerable excess, and thus increasing loss of body heat. This, coupled with the insufficient amount of subcutaneous fat of the premature infant, dangerously impedes the retention of body heat. Dr. Levine showed results of his tests of heat conduction in tissues of infants. These revealed that in spite of inadequate elastic tissues in the capillary walls, the premature infant had adequate heat conduction, better even than that of an adult, and hence that poor control of body heat was not due to a sluggish vasomotor system but to the large surface area and to the poor development of subcutaneous fat.

A study of water balance disclosed that the premature infant has an appreciable lowering in fluid output through the skin and lungs, as compared with the normal infant. A relatively greater burden is thrown on the kidneys. It was further found that the urea clearance is lower in premature infants, and increases with growth. The cause of the lowered urea clearance was found to be the decreased patency of the glomerular capillaries and the decreased development of epithelium of Volkmann's capsules. Impairment of kidney function was thus added to the list of handicaps to be met by the premature infant.

In the gastrointestinal tract, there are the problems of poor sucking, poor secretion of enzymes and, especially, poor absorption of fats. Premature infants were found to excrete more than the normal 5 to 10 calories per kilogram. However, carbohydrates and proteins were found to be metabolized satisfactorily, as evidenced by normal respiratory quotients and normal values for nitrogen absorption. This led to the practical suggestion that, to make premature infants increase weight most rapidly, fats should be replaced by carbohydrate and protein, instead of the usual custom of merely increasing the total number of calories in the diet.

There are several reasons for anemic tendencies in premature infants, such as deficient antenatal iron and copper storage, increased rate of neonatal blood destruction, incomplete development of respiratory enzymes and high incidence of digestive upsets. Hemorrhagic tendencies are present, owing to low prothrombin content of the blood and to weak capillaries, which are weakened further by poor elastic tissue.

There are several rachitic tendencies: reduced calcium and phosphorus storage, which is increased still more by the necessity of keeping premature infants indoors, rapid rate of growth, with a resultant drain on bone and other tissues, and decreased absorption of fats and fat-soluble vitamins.

Finally, Dr. Levine reported that vitamin C had been shown to have a specific effect in protein metabolism. As evidence of incomplete protein metabolism, he was able to demonstrate phenylalanine and tyrosine in the urines of premature infants; these substances were found in larger amounts when cow's milk was being used, because of the relatively high protein content of cow's milk. The oral administration of vitamin C, in the form of 100 to 500 mg. of levoascorbic acid, caused the disappearance of phenylalanine and tyrosine from the urine; hence, he concluded that it is advisable to administer vitamin C to premature infants soon after birth, especially if cow's milk is being used.

BOOK REVIEWS

Strange Malady: The story of allergy. By Warren T. Vaughan, M.D. 8°, cloth, 268 pp., with 23 illustrations. New York: Doubleday, Doran and Company, Incorporated, 1941. $3.00.

Any book that sets out to interpret science for the layman is as difficult to write as it is to review. How much knowledge should the writer expect his public to possess? How is he to present his subject so that it gives

balanced understanding? The reviewer, if he is a specialist in the same field, may quite justifiably think that some things are oversimplified and others not sufficiently explained.

Patients with allergy will get some understanding of their malady from Dr. Vaughan's book. They will not, however, get a balanced point of view. Little space is given to the straightforward routine methods of study and to the simple, easily discovered environmental allergens that are the cause of the majority of symptoms. Perhaps to make the book more interesting, much emphasis is given to the dramatic, the rare and the complex.

There can be few patients so sensitive to nickel that symptoms of contact dermatitis occur from the amount of nickel that might be left in the skin from a needle used to give an injection hypodermically. Nor are there many patients who, having indigestion following the eating of chicken, can be relieved of their discomfort by enemas of chicken broth. Dr. Vaughan describes the case of a patient allergic to grapes who had a rise in blood pressure of thirty to forty points if he drank a Martini cocktail, but a fall in blood pressure when he drank a Scotch highball. Another patient had sick headaches when he ate veal less than six weeks old. And still another was said to be so sensitive to sage pollen that he had hay fever if he ate mutton taken from an animal that had eaten sage.

The most fantastic note of all concerns a sailor who had an eagle tattoed on his chest. Cinnabar, which contains mercury, had been used for the eyes. When the sailor used mercury antiseptics, the eyes of the tattooed eagle were supposed to have become sore and eczematous. Why they were not continuously eczematous if the patient was really sensitive to mercury is not mentioned. And what the mercury antiseptic did to the lesion on which it was used is also not explained. Stories of this type will raise the readers' eyebrows oftener than they will carry conviction.

These examples are not culled from the book to discredit Dr. Vaughan, whose reputation stands on a firm basis, but merely to show how dangerous it is to write a popular work unless one exercises great restraint. The lay reader would get some idea of the allergic processes, but his picture of what an allergist does would not in any sense be a true one. It is a pity that the author's sense of the dramatic so overshadowed his scientific caution. Many of the examples given would be caviar not only to the general public but also to most practicing allergists.

Studies on the Human Thyroid in Iceland. By Júlíus Sigurjónsson. 8°, paper, 130 pp., with 21 tables and 10 figures. Reykjavík: Pretsmidjan Edda H. F., 1940. 6s.

This book will be of interest to those who are concerned with problems of the thyroid gland. The size of the normal thyroid gland forms the subject of one chapter. Figures are given for the length, breadth, thickness and weight of the thyroid gland, with comparative values for Germany and Iceland. The author concludes that the weight of the gland is a most significant feature. It is noted that the average weight of thyroid glands from Iceland and Japan is low.

The chapter concerning the history of iodine and the early use of iodine in the treatment of goiter is excellent. A discussion of methods for iodine analysis in thyroid tissue is included, together with the method that the author employed in his studies. The proportional and total iodine content of thyroid glands from Iceland is compared with that of glands from other districts. The values for Iceland are generally lower and compare with those found in Japan.

According to the author's results, the size of the thyroid gland increased when acute infectious processes had preceded death; in other diseased conditions, there did not appear to be any significant change.

The chapter on hyperplasia of the thyroid gland and its relation to iodine content is the best section of the book. This includes an excellent analysis of present theories, based on available experimental data, and emphasizes the viewpoint that hyperplasia of the thyroid gland indicates a relative iodine insufficiency. Whereas Marine has stated that the iodine content of the thyroid gland per gram weight is the important factor in the presence of colloid or hyperplastic thyroid tissue, the author believes from his studies that the total iodine content of the thyroid gland is a more significant figure. Since, in Iceland, the thyroid glands are smaller, the iodine content is less, and colloid goiter is rare; this difference in viewpoint is obvious.

Surgical Anatomy of the Head and Neck. By John Finch Barnhill, M.D., LL.D., and William J. Mellinger, M.D. Introduction by Paul S. McKibben. Second edition, rearranged and revised. 4°, cloth, 773 pp., with 431 illustrations. Baltimore: Williams and Wilkins Company, 1940. $15.00.

The important change in the second edition of this work consists in the removal of the illustrations from a previous separate section, and their placement throughout the text where they can be consulted with ease.

The book furnishes a good comprehensive survey of the surgical anatomy of the head and neck, particularly from the viewpoint of the otolaryngologist. The material is covered in much greater detail than is usual in texts on general surgical or applied anatomy.

Although the conversational tone of the book, its arrangement about regional dissections and its rough but original drawings prevent the work from being concise, they do make it stimulating and rather charming.

Diagnosis and Treatment of Menstrual Disorders and Sterility. By Charles Mazer, M.D., and S. Leon Israel, M.D. 8°, cloth, 485 pp., with 108 illustrations. New York: Paul B. Hoeber, Incorporated, 1940. $6.50.

It would be difficult to find a more comprehensive book than this on the subject of sterility and endocrine disorders peculiar to women. Not only are the abnormal manifestations of endocrine function as they apply to menstruation fully described and treatment suggested, but at the beginning of the book there are four chapters on the normal endocrine relations. The section on sterility is very complete, and when necessary, the description of the endocrine factors is repeated. In the sterility section, also, there is an interesting chapter that deals with habitual abortion. The diagnosis and treatment of male sterility and the technic of semen examination are included. Other tests, such as the tube test, endometrial biopsy and so forth, that are useful in the diagnosis of sterility are also described in detail.

There are many excellent illustrations and a well-arranged index; a large bibliography is appended to each chapter.

Two criticisms should be made, although neither is particularly serious. The first is that, although a great deal of the material dealt with by the book is highly controversial, the authors tend to be somewhat dogmatic, and anybody using it must realize that the last word has not been said on many of these subjects. The other criticism is that the chapters on male sterility are written by Dr. Charles W. Charny, which seems to indicate that Drs. Mazer and Israel do not themselves check the husbands of their patients. The reviewer is convinced that an adequate picture of sterility in a couple can be obtained only when one man does the work-up on both partners.

In general, however, this book can be recommended highly, although its price will probably keep it off the shelves of anybody but the specialist in this field.

Methods of Treatment. By Logan Clendening, M.D., and Edward H. Hashinger, M.D. Seventh edition. 8°, cloth, 997 pp., with 138 illustrations. St. Louis: The C. V. Mosby Company, 1941. $10.00.

This is the seventh edition of a text on treatment in internal medicine, first published in 1924. It is a summary of therapeutic procedure in one compact volume presented in the concise, lucid style characteristic of its senior author, Logan Clendening. Based both on material gathered discriminately from the ever-changing medical literature and on the wide clinical experience of the collaborating authors, all specialists in their respective fields, this work should be valuable to the general practitioner. The first part is devoted to methods of treatment in all its aspects, the second to their application to particular diseases. The work is well illustrated.

Man's Greatest Victory over Tuberculosis. By J. Arthur Myers, Ph.D., M.D. 4°, cloth, 419 pp., with 31 illustrations. Springfield, Illinois: Charles C Thomas, 1940. $5.00.

This book deals chiefly with the remarkable accomplishments and progress made by veterinarians in the prevention of tuberculosis in cattle. Dr. Myers's main thesis is that a positive reaction to the tuberculin test signifies the presence of tuberculous lesions. Thus he extols the veterinarians for their steady insistence on the tuberculin testing of all herds and the elimination of the positive reactors. That the veterinarians have been successful in their task is attested by the fact that the entire nation is now an accredited area, which means that the number of cattle reacting to tuberculin is less than 0.4 per cent. Whereas in 1917 only 20,000 cattle were tested, 25,000,000 tuberculin tests were carried out in 1935.

This book consists of twenty-six chapters, very well written in a remarkably easy and fluent style. Each chapter has a good summary. The last chapter, entitled "Lessons for Physicians in Human Medicine," is apparently the real reason for the writing of the book.

The author, who has always been in the forefront of the fight against tuberculosis, has been the advocate of segregation and the more aggressive treatment of "open" cases of pulmonary tuberculosis. He has always been the opponent of those recommending immunization against tuberculosis with attenuated organisms (BCG). He stresses the fact that immunization has accomplished nothing so far as the prevention of tuberculosis in cattle is concerned, and that good results have been obtained only after all the positive reactors have been eliminated from the herd. Thus he deplores strongly the facts that physicians have done almost nothing to provide quarantine for tuberculous persons, that they have allowed such persons to enter this country without adequate examination, and also that there is no ban on intercounty or interstate travel.

The author points out that a person reacting to tuberculin has definite tuberculous lesions, and hence has tuberculosis. Just what steps we should take in the prevention or treatment of such cases he omits to state. He apparently considers the preventorium to be of little or no value. He wishes only to direct attention to these matters, leaving their correction to the proper health authorities.

Veterinarians, particularly, should find this book interesting and instructive. In addition, it contains much material that should aid physicians in dealing with the many problems of human tuberculosis.

A Handbook of Elementary Psychobiology and Psychiatry. By Edward G. Billings, M.D. 16°, cloth, 271 pp. New York: The MacMillan Company, 1939. $2.00.

This little book represents an attempt to summarize psychiatry from the psychobiologic point of view. The author has spent some time at the clinic of Dr. Adolf Meyer at Johns Hopkins, has acquired an understanding of his point of view and terminology, and has added to this the experience of working at the Psychiatric Department at the University of Colorado.

The chapters dealing with psychiatric examination procedures and with the descriptive material of the psychotic syndromes are by far the best. Brief and succinct methods are presented for obtaining the complete psychiatric history and mental status. The types of disturbed behavior encountered in psychiatric work are well described. It is in these that the reviewer believes that the major contributions of the psychobiologic school have been made. The emphasis on a good clinical study of the patient and on a careful and helpful observation of the patient is most creditable.

The chapter on psychobiology is an understandable presentation of an important point of view in psychiatry. It is well done, and its limitations are the inherent limitations of that approach and its terminology. The chapter on psychotherapy is clear, but little reference is made to data indicating the therapeutic value of the approaches suggested. Any therapeutic procedure, logical as it may seem, is only of interest and theoretical value unless it is supported by a series of cases proving its validity. The selected readings in Part V represent the basic writings in the field of psychiatry.

This book has value for the student, the internist, the surgeon and the psychiatrist. It fulfills the purpose of the author.

Diseases Transmitted from Animals to Man. By Thomas G. Hull, Ph.D. Second edition. 8°, cloth, 403 pp., with 45 illustrations. Springfield, Illinois: Charles C Thomas, 1941. $5.50.

The nexus of bacteriology and clinical medicine is very adequately represented in the study of diseases common to animals and man. Their manifestations as disease forms, the distinctive immunologic variations and the manner of spread are all important to those seeking knowledge in this field. This volume in a compact form offers a wealth of information of this nature. The list of diseases is too long to enumerate, and each is prefaced by brief historical data of great interest.

The type and format are so outstanding as to merit distinct praise.